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# PRACTICAL PEDIATRICS

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McKEE AND WELLS

VOLUME I







VARICELLA (IN A CHILD OF 8 YEARS)

*Courtesy of Dr. Wm. B. Trimble*

# PRACTICAL PEDIATRICS;

A MODERN CLINICAL GUIDE IN THE DISEASES  
OF INFANTS AND CHILDREN FOR  
THE FAMILY PHYSICIAN

BY

JAMES H. MCKEE, M. D. and WILLIAM H. WELLS, M. D.

PROFESSOR OF PEDIATRICS, IN THE MEDICAL DEPARTMENT OF TEMPLE UNIVERSITY; VISITING PEDIATRICIAN TO THE PHILADELPHIA GENERAL HOSPITAL; GARRETSON HOSPITAL; AND SAMARITAN HOSPITAL, PHILADELPHIA, AND TO THE BANCROFT SCHOOL FOR MENTALLY DEFICIENT CHILDREN AT HADDONFIELD, N. J., FORMERLY PROFESSOR OF PEDIATRICS, IN THE WOMAN'S MEDICAL COLLEGE OF PHILADELPHIA, ETC.

ASSISTANT PROFESSOR OF OBSTETRICS, JEFFERSON MEDICAL COLLEGE; PEDIATRICIAN TO THE WILLINGS' DAY NURSERY; ASSISTANT OBSTETRICIAN, MATERNITY DEPARTMENT, JEFFERSON MEDICAL COLLEGE HOSPITAL; FELLOW OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA. FORMERLY ADJUNCT PROFESSOR OF OBSTETRICS AND DISEASES OF INFANTS IN THE PHILADELPHIA POLYCLINIC AND COLLEGE FOR GRADUATES IN MEDICINE, ETC.

WITH  
AN APPENDIX UPON

## DEVELOPMENT AND ITS ANOMALIES

BY

JOHN MADISON TAYLOR, A. M., M. D.

ASSOCIATE PROFESSOR OF NON-PHARMACEUTIC THERAPEUTICS, MEDICAL DEPARTMENT, TEMPLE UNIVERSITY, PHILADELPHIA. FORMERLY PROFESSOR OF DISEASES OF CHILDREN, PHILADELPHIA POLYCLINIC AND COLLEGE FOR GRADUATES IN MEDICINE; AND OF THE MEDICO-CHIRURGICAL COLLEGE OF PHILADELPHIA; FELLOW OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA; CORRESPONDING MEMBER OF THE SOCIÉTÉ HYGIÉNIQUE DE FRANCE; CONSULTING PHYSICIAN TO THE SCHOOLS FOR FEEBLE MINDED CHILDREN AT ELWYN, PA.; AT VINELAND, N. J.; AND AT HADDONFIELD, N. J.; ETC.

WITH 2 COLORED PLATES  
AND 146 ILLUSTRATIONS INCLUDING 238 FIGURES

IN TWO VOLUMES  
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## PREFACE

This is the Golden Age of the Child. Economists in all lines are now acutely aware that the largest national asset is the sound young human unit. To fulfil this rôle the primary requisite is health of body and mind.

Eugenics is fast becoming (as Galton expressed the fervent hope it would be) an integral part of faith, of "religion," and while parenthood and race culture are being studied earnestly by scientists, yet there is much to learn before eugenics can secure communal encouragement.

Truths emerge slowly from many carefully grouped findings, and then only after much revision and rejection, and by reason of wide and generous co-operation.

No truths can be more worthy of pursuit than those underlying and conditioning the buddings of human life forces. The young human being is too often regarded by the pediatricist, and yet oftener by the pedagogue, as an adult in miniature; ways and means for conservation, repair and education being devised and applied on this wholly erroneous basis. Biologists have made it clear that the young child reaches an acme of vitality during the nursery-age, which demands conservation then and there, or irreparable losses will occur and departures from normal growth begin. Clinicians are reaching the same conclusion in respect to the paramount need for such a prompt and complete recognition and repair of abnormalities and ailments as shall turn aside currents of hurtful influences inducing damagement or hypoplasia.

Thus it will be evident that the subject of developmental abnormalities demands earliest recognition; equally so of the incipience of acquired abnormalities, significant not only in and by themselves, but in the effects they must induce upon organic integrity and growth of the organism in part or in whole.

The child, then, must be studied as a concrete entity, and from all aspects, if we are to do our duty as conservators of health. Moreover, it will be plain that the best economy is to study disease in childhood which is the beginning of research at the center; whereas

in studying disease in the adult, the problem is approached from the periphery or after the plastic age is passed and many irreparable conditions established.

**Practical Pediatrics** is designed to offer the latest word, not only in contemporary findings, but also in the results of the writers' somewhat large clinical experiences on the subject of conservation of infants and children, in and out of health. The authors have endeavored to present succinctly much more than a treatise on disease as it appears in the young; they have kept in the foreground of their purpose a presentation of factors making for, not only the recognition and repair of abnormalities and disturbances, but also prevention in the widest sense of the word.

The writers have endeavored to present many salient points and fundamental principles of development, which they hope will prove of service to practitioners of medicine, and also that the more comprehensible of these points shall be passed on to parents.

The work of the authors throws them in contact with many infants and children, sick and well. Circumstance, also, has cast them into teaching rôles. They realize that pediatrics is growing more rapidly now than the whole subject of medicine grew one hundred years ago, and have been keenly watchful that nothing new should be included that was likely to be discredited.

The present work marks an advance, and it is hoped a significant one, on an earlier publication by two of the authors, who have chosen the third author to lead in the movement of bringing it up to modern needs. The former work (Taylor and Wells) in its two editions was well received and was done into Italian. This translation was at that time an honor unique in works on pediatrics.

Of the newer aids in medicine that on blood-pressure in the young appears for the first time, so far as we know, in a book on Pediatrics. Proper regard has been given to bacterins, tuberculin, and serum treatment. The exact interpretation of the physiological action of drugs has also been attempted, as far as is known, as well as the diagnosis and general principles in the treatment of deformities.

Most of the new illustrations in this work are original (one of us always carrying a camera in his medical grip), but some of them Dr. Taylor has collected, and credit for these is given in their respective legends. In their selection the differentiations of typical conditions have been featured.

The chapters dealing with Fundamental Difference Between Child and Adult, Physical Examination, Infant Feeding, Coryza,

Bronchitis, Pneumonia, Empyema, and Diseases of the Nervous System, have been given special care. Likewise those on Hemorrhagic Diseases, Blood-pressure in Early Life, Hook-worm Disease, Pellagra, and Mental Deficiency. Along with these we must mention the chapters of great interest and value by Dr. Taylor on Development and Its Abnormalities.

With sincere regret, the present authors have seen Dr. Taylor pass from the Pediatric field into other regions of activity and thought, but this regret is tempered by the satisfaction they feel in having him as a contributor.

In making the earlier work the aid of several able specialists was enjoyed. Drs. Arthur W. Watson and G. Hudson Makuen contributed to the chapter on the Nose and Throat; Dr. George C. Stout, on the Ear; Dr. Charles N. Davis, on the Skin; Dr. James H. McKee, on Diseases of the Heart, and Dr. William Johnson Taylor, on many surgical points.

The authors acknowledge with deepest thanks valuable help from Dr. James F. Prendergast on Blood-pressure; Dr. Elon L. Kanaga, on the Development of the Teeth; Dr. Charles P. Noble, on Heredity and Hypoplasia; and an important article, by Dr. Charles E. de M. Sajous, on the Influence of the Ductless Glands on Development.

JAMES HERBERT MCKEE.

WILLIAM HUGHES WELLS.



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# PRACTICAL PEDIATRICS

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## CHAPTER I

### FUNDAMENTAL DIFFERENCES BETWEEN CHILDREN AND ADULTS

Pediatrics, the study of the diseases of infancy and childhood, has been cleverly defined as "the specialty of the general practitioner." In a sense this is true, for more than half of the patients treated in general practice are below the age of puberty. But too often children are not understood, or are thought to be harder to study than adults, simply because the medical attendant, like the ancient Greek pedagogue, tends to view the child as a miniature adult. Nothing could be much further from the truth. As a matter of fact, children differ from adults in three fundamental and essential respects: 1. In their structure (anatomy); 2. in their activities or functions (physiology); 3. in their reactions to disease (pathology). A very few examples will serve to convince any open-minded person of such differences:

1. **Anatomy.**—An adult with a head one-quarter the length of his body; with legs representing but another quarter, would indeed appear a monstrosity. Yet such proportions are normal to the infant at birth. And what is true of differences in general structure is true of almost any organs or tissues that we examine. More than this, it is true of microscopic as well as macroscopic appearances.

2. **Physiology.**—A baby's inactivity, his weakness as we say, is in striking contrast to the coordinated muscular activity of the adult. Differences almost as striking exist between the digestive functions at these different ages. And so, in almost every sphere of bodily activity, similar age differences could be found.

3. **Pathology.**—The diseases of early life are either peculiar to certain ages, or else are morbid processes so modified by their occurrence in the immature organism that they appear more or less different

from the same diseases as seen in the adult. Thus rickets is a characteristic disease of infancy, rarely occurring in later childhood, and never in the adult. Scurvy, on the other hand, may occur at any period of life; but he who knows scurvy only as it appears in the jail or the barrack room, etc.; would scarcely recognize as such the Barlow's disease of infancy. And so, once more, we might continue to multiply examples.

Before we may hope to understand children, there is another important conception that must be grasped: The child at whatever age we find him represents an unstable organism, constantly undergoing growth and other changes. Nor are these changes progressing at the same rate in season and out, in this organ and in that; far from it. The body as a whole grows more rapidly at some ages than at others, and one organ may be advancing rapidly in size, while another remains stationary or actually diminishes.

With these fundamental facts in mind we shall immediately proceed to a study of

## THE PHYSIOLOGY OF THE INFANT AND CHILD

### SIZE AND WEIGHT

A fully developed infant is one born after a period of two hundred and eighty days' gestation. The average length at birth is from seventeen to twenty inches, or about fifty centimeters, and the weight, as given by different authorities, varies from six to eight pounds (averaging about 4000 gm.). The weight of males is slightly in excess of that of females.

The weight of the infant and child, although subject to slight variations due to many causes, should increase in a regular, definite proportion according to the age. Sudden decrease in weight almost certainly points to some fault in nutrition, the use of improper foods, or the approaching onset of some disease.

The average infant will double its initial weight between the fifth and sixth months, and will treble it at about fifteen months. At seven years the weight is double that of one year, and at the fourteenth year it is double that of seven years. It has been pointed out by Proscher that the growth of the infant during the period of suckling increases in proportion to the richness of the mother's milk in proteids and salts, and that this law applies to other animals as well as to man, the young of those animals whose milk is especially rich in proteids and salts increasing in weight more rapidly than others.

He states that an infant fed on mother's milk with a proper per cent. of proteids should double its initial weight in one hundred and eighty days.

The following table will show the gain in weight in grams and pounds from birth to fourteen years:

Age	Grams	Pounds	Average gain a day	
			Grams	Ounces
At birth.....	3,000 to 4,000	6.6 to 8.8		
From birth to five months.....			20 to 30	$\frac{2}{3}$ to 1
From five months to twelve months.....			10 to 20	$\frac{1}{3}$ to $\frac{2}{3}$
At one year.....	9,500	20.9		
At seven years.....	19,000	41.8		
At fourteen years.....	38,000	83.6		

The above table is computed on a basis of 3500 gm. (7.7 pounds) at birth, and of a gain of 30 gm. a day for the first four months and 10 gm. a day for the last eight months of the first year (Rotch).

Boys		Age	Girls	
Height, inches	Weight, pounds		Height, inches	Weight, pounds
19.75	7.15	Birth.	19.25	6.93
24.75	14.3	5 months.	23.25	13.86
29.53	20.98	1 year.	29.67	19.8
33.82	30.36	2 years.	32.94	29.28
37.06	34.98	3 years.	36.31	33.15
39.31	37.99	4 years.	38.8	36.36
41.57	41	5 years.	41.29	39.57
43.75	45.07	6 years.	43.35	43.18
45.74	48.97	7 years.	45.52	47.3
47.76	53.81	8 years.	47.58	51.56
49.69	59	9 years.	49.37	57
51.68	65.16	10 years.	51.34	62.23
53.33	70.04	11 years.	53.42	68.7
55.11	76.75	12 years.	55.88	78.16
57.21	84.67	13 years.	58.16	88.46
59.88	94.49	14 years.	59.94	98.23

During the first six months the increase in length of the average infant is from 4 to 5 inches; in the second half of its first year the increase is from 3 to 4 inches; during the second year the gain is from 3 to 5 inches; during the third year, from 2 to 3  $1\frac{1}{2}$  inches; and in the fourth year, from 2 to 3 inches. From this time on the increase in length averages about from  $1\frac{2}{3}$  to 2 inches every year. The length of the child at any given age should bear some sort of relation to its weight; this is shown in the second table on page 3.

Slow or arrested growth, if continuous, is always a sign of some pathologic condition. The most common of these is malassimilation of food, but more serious conditions, such as rickets or syphilis, must be considered. Arrested growth is very commonly found in children affected with chronic diseases of the brain and in cretins.

In infancy the tissues are softer and more elastic than those of the adult or even of older children. The body and limbs are well rounded by a plentiful covering of fat, giving the characteristic plumpness and roundness so familiar in the infantile form. A downy growth, known as lanugo, frequently covers the body at this time; the nails are well formed, and extend to the end of the pulp of the fingers and toes. A glance at the body as a whole will at once reveal the fact that the upper part of the trunk, with the arms and head, is much larger than the lower part of the body, with the exception of the abdomen. The cause of this will easily be seen when we study the changes in the circulation of the blood which follow the cessation of placental and the establishment of pulmonary circulation. Observations made on a large number of infants show us that certain parts of the infant's body must attain certain definite relations to one another in regard to size, in order to show that it has attained a perfect state of development. A short rule for computing this is the following: As has been before stated, the length of the average child at birth is about 20 inches, or 50 cm. If the infant is normally developed, the circumference of the thorax should measure one-half the entire length of the body plus 10 (50 divided by 2 equals 25; plus 10, equals 35 cm.). Therefore 35 cm. is the circumference of the thorax in the normal infant. The circumference of the skull should equal the circumference of the thorax plus 2, or 37 cm. (50 divided by 2, plus 10, equals 35; plus 2, equals 37 cm.). This simple rule is of practical value, and has been tested by the authors in a number of cases. According to Frank, who has made a series of studies on the relative size of the head and of the shoulders of infants born at term, the girth of the shoulders is usually greater than that of the head. When the circumference of the head

falls below 32 cm. (12 5/8 inches), the child is usually immature. The growth of the finger-nails, the presence of lanugo, and the size of the epiphyses he considers of little importance as diagnostic signs of the maturity of the fetus. Changes in the proportions given above in a majority of cases will point to some abnormality in the development of the child or to the presence of disease; thus, when the circumference of the head greatly exceeds its relative proportion to the thorax and length of the infant, the patient is either rachitic or, more probably, has beginning hydrocephalus. A considerable decrease in these proportions would very probably indicate microcephalus, and if the fontanelles are absent or very small, the diagnosis would be almost certain.

An examination of the trunk and extremities of the new-born infant will reveal the fact that the former is ovoid in shape with the larger end below, the greater part of this lower end being taken up by the abdomen, which at this period of life is large in proportion to the rest of the body. The reason for this will be explained when we study the proportionate size of the various abdominal organs, especially the liver, which during fetal life and infancy is of a size out of all proportion to the other abdominal organs. The pelvis and lower extremities are proportionately small and ill developed, while the upper extremities "appear as small, jointed outgrowths from the smaller end of the ovoid trunk."

Usually the first spontaneous act of the new-born infant is a lusty cry. By this means air is inspired into the lungs, which expand for the first time, and thus aid in establishing pulmonary respiration. When nude and lying on its back, we see the new-born infant making almost continuous movements with the legs and arms, accompanied by a certain amount of flexion and extension of the spine. These movements are probably a continuation of those made in the uterus during fetal life. When asleep, the attitude is normally one of complete repose, there being no motion except that of respiration, the type of which may vary considerably even in health.

### TEMPERATURE

At birth the rectal temperature of the infant is about 100° F. (37.5° C.), the axillary temperature being about two degrees lower. According to some authorities, a slight fall of two or three degrees occurs a short time after birth; this, however, in the course of a few hours rises to 98.5° F., which is the normal temperature of the human body. Rotch gives the temperature of the new-born as follows:

At birth.....	37.2° C. (99° F.)
Within an hour.....	36.1° to 35.5° C. (97° to 96° F.)
In about a week.....	36.8° C. (98.2° F.).

There is, however, in all cases a certain amount of variation which must be considered to be within the normal limit. This normal variation is considerably greater in infants than in adults, with the exception of the temperature-ranges seen in women during the puerperal period. In premature infants or in those whose development is under the average at birth the temperature is apt to be below the figures which have been given. Reitz and Finlayson have shown that there is a slight variation in temperature at different times in the day: the highest point being between 5 and 6 P.M., and the lowest occurring during the early morning hours, say between 4 and 5 A.M. It is of importance to remember that in young children trifling causes may often produce considerable increase in bodily heat. An example of this is seen in the fever which accompanies various slight nutritive disturbances.

### CIRCULATION

Previous to birth, the course of the circulation in the fetus is as follows: The fetal blood, separate and distinct from the maternal, flows from the placenta through the umbilical vein and enters the body at the umbilicus, passing thence directly to the liver. At this point a division in the current takes place, part of it passing through the ductus venosus to the inferior or ascending vena cava, the remaining portion going directly into the portal vein and passing through the liver; it then enters the ascending vena cava, whence it goes into the heart at the right auricle. Here the blood currents from the superior and the inferior vena cava empty, but do not unite, and instead of passing into the right ventricle, as is the case in adults, the blood is directed by the Eustachian valve into the left auricle through the opening known as the foramen ovale. Thence it passes into the left ventricle, and so into the aorta, whence it is finally distributed through the general system. The greatest amount of blood containing the most oxygen is carried by way of the carotid and subclavian arteries to the head and upper extremities; this accounts for the great development of the latter at birth. Returning from the upper part of the body, the venous blood is carried along the superior vena cava into the right auricle, from which it passes through the tricuspid valve into the right ventricle, and thence into the pulmonary arteries in the same

manner as in the adult. From the pulmonary artery a small quantity of blood is allowed to pass through the lungs, but the greatest portion of it is carried through a tube which, during fetal life, connects the pulmonary artery with the aorta, and which is known as the *ductus arteriosus*. By this avenue it is carried into the aorta. The amount of blood which passes directly through the pulmonary arteries into the lungs is only sufficient for their nourishment, and the return circulation then passes, as in the adult, through the pulmonary veins entering the left auricle, where it mingles with the blood, passing by way of the foramen ovale, and thus enters the aorta. Subsequently the blood, laden with the excrementitious substances of the fetus, is carried back to the placenta through the umbilical arteries. These are continuations of the right and left hypogastric arteries which arise from the internal iliac arteries. The changes in the circulation following birth and the cutting-off of the placental circulation are as follows: (1) Expansion of the lungs produces, by degrees, a closure of the foramen ovale, thus relieving aortic pressure and producing obstruction of the ductus arteriosus, which gradually degenerates into a fibrous cord. At times neither the closure of the foramen ovale nor of the ductus arteriosus occurs immediately after birth; indeed, the foramen ovale may remain open for a month and the ductus arteriosus for a considerably longer time. (2) The umbilical vein and the ductus venosus become obliterated, usually from the second to the fifth day after birth; ultimately they degenerate and become fibrous cords, the former becoming the round ligament of the liver and the latter a fibrous cord, which in the adult may be traced along the fissure of the ductus venosus. (3) The umbilical arteries change in a twofold manner; the portion between the internal iliac and the superior vesical branch, which supplies the bladder, remains pervious, while the portion between the fundus of the bladder and the umbilicus becomes obliterated—usually from the second to the fifth day—and continues as a fibrous cord which forms the lateral ligaments of the bladder. (4) The fetal function of the Eustachian valve ceases, almost immediately after birth. Its remains, however, may be traced for weeks or months afterward. In the new-born infant the weight of the blood is estimated at 5 per cent. ( $\frac{1}{19}$  to  $\frac{1}{20}$ ) of the entire body weight, while in the adult it is about 8 per cent. ( $\frac{1}{13}$ ) of the body weight. The red corpuscles at birth may be found in greater number than in the adult. In a few days this number decreases appreciably. The hemoglobin curve closely follows the red blood cells. Red corpuscles vary in size more than they do in the adult; and nucleated forms are occasionally found.

The number of leukocytes is greater than in the adult and this relative disparity is observed in lessening until late in childhood. Comparative counts of the white cells, also reveal that the lymphocytes are much more numerous in infancy and childhood than they are in later years (see Diseases of the Blood). Indeed it has been well said that if the blood picture of an infant were to be presented by an adult's blood, the latter would be viewed as distinctly pathologic.

### HEART

In its early stage of development the heart is so large as to occupy the greater part of the thoracic cavity. According to McClellan, it is at this time much larger, proportionately, than at any later period, or even subsequent to birth. The relative size of its cavities is also different at this time, the auricles being considerably larger than the ventricles, and the right auricle being the larger of the two. As the organ progresses in its development we find the ventricles gradually equal and then exceed the auricles in size. The peculiarities of structure of the fetal heart have already been dwelt upon under the head of fetal circulation; they may, however, be summed up briefly in the following: Between the right and the left auricle we find an oval opening, known as the foramen ovale, which allows the passage of the blood between the two auricles; also the Eustachian valve, which directs the blood coming from the inferior vena cava through the foramen ovale.

After birth and subsequent to the commencement of pulmonary respiration, with the complete establishment of the function and structure of the lungs, we find these organs filling out their natural space in the thorax, and after this the heart attains nearly the proportionate size to the other organs that it does in the adult, and its position will be found, by external examination, to be very nearly the same. It is not, however, covered by the lungs to so great an extent as in the adult, and this difference is caused, partly, by the presence of the thymus gland. External examination during infancy will show that the cardiac impulse is higher and is found to the left of the mammary line, not inside of it, as in adult life. The apex beat is often hard to detect in infancy. The apical impulse does not occupy its adult position until the fourth year (Steffen) or even later. Prior to this period it is usually felt in the fourth interspace.

Later in childhood the apex can be made out with great clearness; in fact, is often more distinct than in the adult. An accurate examination of the valves of the heart is difficult, particularly in early childhood, partly from the presence of the thymus and partly from

the fact of the high position of the heart in the chest causing confusion of the sounds of its valves with those of respiration. It is pos-



FIG. 1.—DISSECTION OF A NEW-BORN INFANT, SHOWING THE SIZE AND POSITION OF THE THYMUS GLAND AND ITS RELATION TO THE LUNGS.

A. Thyroid gland. B. Thymus gland. C, C. Lungs. D. Esophagus. E. Stomach. Natural size. Dissection made by Dr. F. S. Ferris from a case in his own practice.

sible, also, that the heart may change its position with the movements of the body to a greater extent in childhood than in later life.

The heart-beat in the new-born infant varies from 120 to 140 pulsations a minute, girls having a slightly more rapid heart action than boys. According to McClellan, the pulse-rates in subsequent years are as follows:

In the second year, 100 to 115 beats a minute. From the seventh to the fourteenth year, 80 to 90 beats a minute, and after that, 75 to 80. It must be remembered, however, that the normal rate and the rhythm may both be greatly changed by slight causes.

The weight of the heart at birth is about 20.5 gm., or about two-thirds of an ounce.

## LUNGS

The apices of the lungs in children are found by external examination to occupy almost the same position as in the adult: that is, between one and two fingerbreadths above the clavicle. The vesicular sounds are most distinct below the clavicle, while the bronchial sounds are best heard in the upper region of the sternum. Owing to the encroachment of the liver upon the right side of the thorax, a considerable difference is found in the size of the spaces occupied by the right and the left lung, the right lung extending downward as low as the eleventh rib posteriorly, while the lower border of the left lung is found as low as the twelfth rib. Anteriorly, the right lung extends to the fourth or fifth rib, while the lower border of the left lung is found at the margin of the sixth rib. The bronchial portion of the respiratory tract is much more highly developed than the vesicular during early infancy, the vesicular portion consisting merely of small bud-like dilatations at the ends of the lesser bronchi. The connective-tissue element is also found in greater abundance in infancy than in later childhood or in adolescence. The blood-vessels of the lungs are more distensible and tortuous during early childhood than in later life. The air capacity of the lungs is smaller proportionately during early infancy than in late childhood or adult life; however, we find this increasing rapidly as age advances. According to Schnepf and Wintrich, the vital cubic capacity at different ages is:

Three to four years, about.....	450 c.c.
Five to seven years, about.....	900 c.c.
Eight to ten years, about.....	1,300 c.c.
Eleven to twelve years, about.....	1,800 c.c.
Thirteen to fourteen years, about.....	2,200 c.c.
In adults (average), about.....	3,300 c.c.

The absorption of oxygen, however, is relatively greater during childhood than in adult life, while the expiration of carbonic acid gas is somewhat less. In new-born infants the number of respirations varies from about forty to forty-five a minute; from this number they slowly decrease until the third or fourth year, when they number about from twenty-five to thirty. At the beginning of adolescence, or about the fifteenth or sixteenth year, they average twenty. The type of respiration in the infant is always abdominal, for the reason that during early life the diaphragm is the largest factor in the production of respiration, the ribs moving but slightly and the abdominal muscles aiding but little in the respiratory movements. Any alteration of this type of respiration is nearly always a symptom of disease in a young child. In later childhood, however, we see the chest participating in the act of respiration, the ribs being raised by the action of the other respiratory muscles or the thoracic muscles of respiration. The action of the intercostal muscles is of slight importance in man. As has been pointed out by Ebner and others, the muscles of inspiration, aside from the diaphragm, are the quadratus lumborum, the serratus posticus inferioris, the serratus posticus superioris, the levatores, the scaleni, etc. The intercostal muscles are chiefly of importance in giving rigidity to the chest. As the child approaches puberty we see the type of respiration varying with the sexes, the abdominal and inferior costal type being found in males and the superior costal type (so called) in females. In girls there is much less movement of the abdomen, while the greater amount of respiratory movement is seen in the upper part of the thorax. The rhythm of respiration may vary considerably in children, especially in the very young. It may, in fact, become irregular without indicating disease. The costal pleura in children is somewhat thicker than in adults, and the inferior margin of the pleura may extend as low as the articulation of the twelfth rib, and sometimes even as low as the transverse process of the first lumbar vertebra posteriorly.

### THORAX

The thorax in the infant and young child is of much less consequence as a part of respiratory mechanism than it is in the adult. During early life it is somewhat narrower proportionately in its transverse diameter than is the thorax of the adult. The average antero-posterior diameter of the interior of the adult thorax bears a relation to the transverse of 1 to 2  $\frac{1}{2}$  or 3. In the infant at birth this relation

bears a proportion of as 2 is to 3 (Rotch). The thorax is also shorter proportionately from above downward in the infant than in the adult. The sternum in the young child is placed higher than in the adult, the top of the manubrium being on a level with the first dorsal vertebra in the child, while in the adult it reaches to between the second and third vertebræ. In children with rickets the manubrium not infrequently reaches as high as the thyroid cartilages, as in a case seen in Dr. Taylor's clinic. The shortness of the neck so produced may have important bearing on the operation of tracheotomy and other surgical procedures about the neck. The shape of the sternum during early childhood is said to approach more nearly that of the female than the male. The ribs, too, are more nearly horizontal, and form a larger part of the lateral walls of the thorax during childhood than in adult life. In quite a number of cases a certain amount of variability may be found in the development of the thorax. Especially is this the case in the measurement of the two sides, the circumference of the right side often being greater than that of the left.

The ribs of the child are flatter and more elastic than are those of the adult, but, owing to the inferior development of the inspiratory muscles, they are raised but slightly during respiration, unless the breathing is forced. As has been already pointed out, respiration in the child is chiefly accomplished by the action of the diaphragm, so that the movement of the thorax is comparatively small. The bones of the thorax are subject to various deformities, caused by the influence of disease. Thus, in rickets we may have a formation of bony protuberances at the junction of the ribs and the costal cartilages, or from extreme softness of the structure there arises a narrowing of the chest with a protuberance of the sternum, causing the deformity known as *pigeon-breast*.

#### DIAPHRAGM

The diaphragm occupies a higher position in children than in adults, and is proportionately better developed on account of its importance as an organ of respiration. In regard to its position, Rotch, basing his opinion largely on the observations which Dwight made upon frozen sections, states that in the infant the diaphragm is to be found opposite the eighth and ninth dorsal vertebræ. On the right side it rises higher than on the left, arching over the liver. The lowest portion is that extending along the central or median line. Anteriorly, in the young child it is said to be inserted somewhat above the apex of the ensiform cartilage.

## ABDOMEN

The abdomen of the child is more protuberant and is of a larger size relatively than that of the adult, owing, chiefly, to the greater size of the liver during early life. This organ, as has already been explained, occupies almost the entire right side of the abdomen, and, according to McClellan, has a relative weight to the whole body at birth of about 1 to 8. Its lower border, with the child in the upright posture, reaches nearly to the crest of the ilium, and its left lobe\* extends across to the costal cartilages of the left lower ribs. In the middle line the liver is in close relation to the skin in front of the stomach, and reaches half way between the ensiform cartilage and the umbilicus. Its lower edge corresponds to a line drawn from the ninth costal cartilage on the right side, to the eighth costal cartilage on the left.

## INTERNAL SECRETIONS AND THE DUCTLESS GLANDS

When, perforce of metabolic activities, glandular cells take up certain constituents of the lymph and blood, and elaborating them in an altered or unaltered state pass them on into their gland ducts, then we have what physiologists call *secretions*. If the secretions are of no further use to the economy, or to offspring (as in the case of milk), they are oftentimes styled *excretions*. If the substances elaborated are recontributed to the lymph and blood, there to perform important offices for the economy at large, one speaks of these substances as *internal secretions*. In its function of internal secretion a gland may be likened to a citizen of a community, who having shared certain communal advantages, contributes his taxes for the maintenance of the whole. In a strict sense all tissues yield such useful substances. The kidney's main office is to secrete (excrete) urine, but it also contributes an important internal secretion. The testicle and ovary also have specific functions (the secretion of semen and the formation of ova), but in addition to these they contribute very important substances to the lymph which are of very great value to the economy.

In recent years, however, it has been found that the so-called ductless glands are particularly active and important in this office of internal secretion. Their products are of vast importance to the body at large in stimulating developmental changes and in maintaining life and health. The ductless glands are the thyroid and parathyroid structures, the thymus gland, the spleen, the anterior lobe of the pituitary body, the suprarenal glands, the lymph nodes and the red marrow of bones.

### THE THYROID GLAND

The thyroid gland is relatively large in the infant and child, according to some as large in the infant as in the adult. Its position relative to other structures in the neck is also higher. The isthmus usually covers the second and third tracheal rings—an important matter to bear in mind in the performance of tracheotomy. Outside of the gland capsule, the parathyroid structures are found.

Many years ago, Schiff noted that the removal of the thyroid gland in animals produced symptoms suggesting the cachexia strumaprima, and in dogs, that death soon resulted with symptoms of tetany. These symptoms were prevented by the implantation of a thyroid gland into the peritoneal sac of the animal operated upon. Later on, the relation of disease (such as goiter) or absence of the gland to cretinism and infantile myxedema became well established. Again it was found that if the thyroid gland of another animal (such as the sheep) was fed to the affected subject, the myxedema began to disappear and the subject to develop in body and mind. At a still later period it was discovered that experimental removal of the thyroid gland without injury to its capsule, thus sparing the parathyroid bodies, did not result in the death of the animal.

There is no doubt but that this body (or bodies) supplies to the organism an internal secretion that is of vast value in influencing bodily development and in the maintenance of bodily health.

With this knowledge at hand, it is small wonder that much attention has been attracted to this structure and its function. Our knowledge concerning both has made a marked effect upon therapeutics and has possibly initiated a new era in treatment.

### THE THYMUS

This structure lies in the anterior mediastinum of the infant and young child immediately under the manubrium sterni and possibly a portion of the gladiolus. It covers the pericardium to a considerable extent. It is a long, lobulated gland, that closely resembles the salivary glands or the pancreas in structure. It has no duct, however. According to some authorities, it attains its greatest size at about the fourth year of life; according to others, as early as the second. In Dwight's frozen sections of a three-year-old child it is beautifully shown, extending as low as the fourth dorsal vertebra. According to Rüdinger it does not atrophy markedly until the fourteenth year; though some

authorities place the period of atrophy at a much earlier time of life. Somewhere between the pubertal period and early adult life, it disappears and is replaced by fatty tissue.

The function of the gland is not positively known; but there is considerable evidence to show that it bears some relationship to the development of the intellectual faculties. In imbeciles it is often lacking (Bournenville). Some physiologists place it among the blood-making organs.

From the clinical side the structure is of considerable interest because of the so-called thymic deaths. Thymic deaths are probably of two varieties: The thymus may be enlarged in the *status lymphaticus* and death may result (probably toxic), or the enlarged gland in other cases may play a mechanical rôle, causing thymic asthma and death. In one case, Chevalier Jackson exposed the gland and found that when he lifted it away from the trachea the symptoms disappeared. This would seem to prove positively that the congested and enlarged gland may play a malign mechanical part.

## SPLEEN

In the child the spleen can be outlined externally on the left side in the neighborhood of the tenth and eleventh ribs, at which point it is nearest the surface of the body. It must be remembered that the diaphragm always intervenes between the abdominal wall and the spleen itself. In examining the organ percussion may prove of little value, as a distended splenic flexure of the colon may interfere seriously with our study, unless very light percussion is employed. Anyone used to examining children should be able to palpate an enlarged spleen, however. When the spleen can be felt beneath the costal border, we may safely say it is enlarged. The size of the spleen is said to vary greatly, according to the state of nutrition, being much larger in well-nourished children than in those affected by wasting diseases. An exception to this will appear in malnutrition due to syphilis, in which case the spleen is frequently enlarged. This obtains even more often in rickets. The size of the spleen is increased during and after digestion, at which time it contains a large amount of blood. It is covered and sustained by the peritoneum. In front of it are the stomach and the splenic flexure of the colon. When enlarged, it is said that the spleen in childhood encroaches more upon the thoracic cavity than in the adult, this being caused by the greater resistance of the costocolic fold of the peritoneum upon which it rests. That the spleen acts as a physiologic

filter for the blood is pretty well proved. It is also a hematopoietic organ, leukocytes, at least, being formed in it.

### THE ANTERIOR LOBE OF THE PITUITARY BODY

The pituitary body has been of interest to anatomists for a long period of time. There has also been much speculation concerning its possible functions. It is probably an important structure physiologically; for it has been found much enlarged in cases of giantism (gigantism) and akromegaly. Sajous believes that it is connected through the sympathetic nervous system with the suprarenal glands, and has expounded an interesting theory concerning its functions.

### THE SUPRARENAL CAPSULES—ADRENALS

There is a period of life when these glands are actually larger than the kidneys. In the new-born, they bear a volumetric relationship of 1-3, when compared to the renal structures; whereas in adult life, the ratio is 1:28. McClellan describes the suprarenal capsules at birth as not only surmounting the kidneys, but as quite covering them.

The exact embryologic derivation of these structures is still a discussed subject. The majority of authorities believe that the cortex of the gland is derived from the pronephron, while the medullary substance, so closely associated with the sympathetic nervous system, is derived from the epiblast. Some investigators doubt the origin of the cortex, claiming that the adrenal structures may be present where the whole urinary tract is absent.

The internal secretion of the glands, styled adrenalin by its discoverer, Takamine, is of vast importance in maintaining the proper degree of tone (vaso-constriction) of the blood-vessels. Sajous, who suggests the name adrenoxin for this substance, claims that it is the oxidizing agent in hemoglobin. This internal secretion is said to arise from the medullary substance. The cortex is said to possess antitoxic properties.

### THE LYMPH NODES

The lymphatic glands are of much interest to pediatricians, for clinically they show a tendency to enlarge upon relatively slight provocation in the infant and child, and such enlargements may prove of great diagnostic import. We shall not go into a description of the

various chains of glands nor of their histologic structures. For these the students are referred to standard works upon anatomy.

Leukocytes are unquestionably produced in the lymph nodes of the body. These structures are also important filters for the lymphatic system. They stand like sentinels at various portals for infection. It is in this latter relationship that the student of the child should ever view them. Thus with a tonsillitis, the tonsillar gland at the angle of the mandible enlarges. When absorption has occurred from adenoid growths of the nasopharynx, the posterior cervical glands are found to enlarge. With invasion of the pulmonary apex (usually with the tubercle bacillus) the supraclavicular nodes may be found tumefied. In general infections, a generalized adenopathy may exist.

### THE RED MARROW OF BONES

It is to be remembered that in early life, practically all of the bony marrow is red marrow. This substance is not confined to the ends of the long bones as in later life. Red corpuscles are produced in the red marrow as are also certain white forms.

### PANCREAS

Although the pancreas is formed about the second month of intra-uterine life, the starch-digesting (amylolytic) action of the pancreatic secretion is not very active until about the fourth or fifth month after birth, and is probably not perfected until the eighth or ninth month. The steatolitic and proteolytic properties, however, are fairly well developed at birth. This has an important bearing on the question of the proper articles of food to be used in early infancy. The relation of this organ to carbohydrate metabolism is probably an important one (see Diabetes).

### KIDNEYS

In new-born infants and young children the kidneys are relatively larger in size and are situated lower than in adults. The latter peculiarity may be explained when we know that the lumbar part of the spine is relatively small at birth. The kidneys lie behind the peritoneum in a considerable quantity of fatty tissue, which helps to hold them in position. Owing to the large size of the liver, the right kidney is situated lower than the left. The kidney in children is more lobulated than in

the adult, and the suprarenal capsules are much larger—indeed, in young infants they sometimes almost cover the kidney.

During the first year or two of life the kidneys attain nearly the same position and relations as in the adult.

The *bladder* in the child is practically an abdominal organ. When distended, it occupies nearly the whole of the lower portion of the abdomen, and this is an important fact to be remembered in making an examination of the abdominal organs of a child. Before such an examination the bladder must always be emptied.

The shape of the bladder is ovoid, with the larger end resting in the pelvis. As the pelvis increases in size the bladder becomes more and more accommodated within its cavity, finally becoming one of the pelvic organs. In the child the peritoneum is reflected entirely over the posterior surface of the bladder, and extends to behind the urachus downward to the neck of the bladder, and thence to the upper part of the rectum. The anterior surface of the bladder is always *free from* peritoneum. The bladder is capable of great distention; cases have been reported where the summit of the organ has reached the umbilicus, and even extended to the ensiform cartilage.<sup>1</sup> The *prostatic gland* is very small during early life.

The *quantity of urine* secreted increases rapidly for the first five days after birth. From that time the increase is slow. The average amount excreted by a child of four or five days is about twelve to fifteen ounces, averaging 420 c.c. At two years of age the daily excretion will average fifteen ounces, or 500 c.c., and at four years eighteen to twenty ounces, averaging about 600 c.c. According to most authorities, the urine at birth is more concentrated, and therefore of a higher specific gravity, than that secreted after the infant has begun to feed from the breast; it will, however, vary considerably with the amount of water the child is allowed to drink. As a rule, however, the specific gravity is lower in infancy and early childhood than in adult life. It may vary from 1000 to 1010. It is curious to note that in the kidneys at birth there is often a peculiar reddish discoloration of the papilla, which is caused by the presence of uric acid crystals. It would seem as if this deposit was particularly marked in those children who have not had a normal supply of oxygen at birth. During early life the urine may be turbid and dark, but later it becomes clear and of a light-yellow color. The excretion of urea is relatively smaller in children, and the same is also true of the chlorids and phos-

<sup>1</sup> In a case reported by Dr. J. M. Taylor the upper surface of the organ reached as high as the diaphragm.

phates. Occasionally traces of albumin may be found in the urine during the first few days of life, but these will soon disappear.

## STOMACH

The stomach is placed more vertically in the infant than in the adult; in fact, its axis is more nearly vertical than horizontal or transverse. Its shape at birth is more tubular than in later childhood or in the adult. As is well known, the cardiac orifice of the fully developed adult stomach is protected by a valve which prevents the regurgitation of fluids into the esophagus. In infants, however, this valvular constriction is deficient, and it is owing to this and to the vertical position of the stomach that regurgitation of fluids so much more frequently occurs in infants. Indeed, this is nature's way of relieving overdistention. At about the middle of the first year of life the stomach is relatively broader than at any later period; the fundus is, at this time, but slightly developed. The capacity of the stomach at birth is very small, nearly all authorities giving it as being about  $4/5$  of an ounce. The size and capacity of the organ seem to vary somewhat with the weight of the child. During infancy the stomach is capable of considerable distention, and it has been found that the stomachs of babies fed continuously on artificial food have a greater capacity at any given age than those fed by the breast. On the other hand, it seems probable that the use of too small quantities of food has a tendency to cause contraction of the stomach, thereby decreasing its capacity. Following birth the growth of the stomach is quite rapid for the first three months of life. This is followed by a period of about two months in which the increase in the gastric capacity is slight. After this time, however, there is a regular and steady increase in size until adult life is attained.

According to Rotch, the increase of gastric capacity is as follows:

3 hours old, capacity	$5/6-1$ oz.	25-30 c.c.
4 weeks old, capacity	$2\ 1/2$ oz.	75 c.c.
8 weeks old, capacity	$3\ 1/5$ oz.	96 c.c.
12 weeks old, capacity	$3\ 1/3$ oz.	100 c.c.
16 weeks old, capacity	$3\ 14/25$ oz.	107 c.c.
20 weeks old, capacity	$3\ 3/5$ oz.	108 c.c.

An accurate study of the capacity of the stomach of infants is of great practical value in aiding the physician to regulate the quantity of food which should be given. As the experience of those interested in the scientific feeding of infants increases, the necessity for such accurate knowledge becomes of more and more importance.

As an organ of digestion, the stomach does not play so important a part in infancy as in adult life. This is said to be owing partly to its more perpendicular position in the infant, and also to the fact that at this period of life the gastric juice is but scantily secreted. When milk is taken, the curd is quickly coagulated by the rennet ferment—this taking from ten to fifteen minutes—after which it is acted on by the pepsin and hydrochloric acid, but before digestion is complete a large portion is passed into the intestine, where digestion is completed. According to Holt, a large portion of the milk passes from the stomach into the intestine in young infants during the first half hour, and at the end of an hour the stomach is empty.

The duration of gastric digestion varies with the age of the infant and the food given; as a rule, human milk is more quickly digested than cow's milk.

The gastric juice of the infant contains pepsin and hydrochloric acid as in the adult, and lactic acid is also occasionally found, but all these elements, and especially free hydrochloric acid, are in much smaller proportion during infancy, and it is supposed that this scanty secretion of hydrochloric acid probably explains the feeble germicidal properties of the gastric juice of infants and the susceptibility of these patients to gastro-enteric infection.

#### SMALL INTESTINE

The average length of the small intestine in the infant at birth is 9 1/2 feet. According to Rotch's measurement, it is 287 cm., although he states that he has seen a variation of 61 cm., or about two feet. During the first month of life this increase in length is about the same, but after that time its growth is irregular. During childhood the upper part of the small intestine usually occupies the left iliac fossa, while the lower part is found in the iliac fossa of the right side. In the first part of the duodenum the glands of Brunner are placed, while in the portion below the duodenum the patches of Peyer can be found, often at a very early period. In the small intestine the process of digestion is continued by the action of the pancreatic juice and other secretions. The fat of the food is here emulsified, and absorption of elements whose digestion was begun in the stomach takes place. It is highly probable that neither digestion nor absorption in the small intestine are the simple processes that physiologists thought them a few years ago. The *secretin* absorbed from the upper portion of the small intestine probably stimulates the secretion of the pancreas very mark-

edly (see Atrophy). In their passage through the intestinal wall the products of proteid digestion are also modified considerably. The ferment of the intestinal wall has been styled erepsin. During absorption, the proteid products before mentioned are broken up further into amino- or amido-acids, and some of these, in turn, are reconstructed into the proteid molecules that appear in the lymph and blood.

## LARGE INTESTINE

At birth, according to Treves, the large intestine measures about one foot, ten inches, or fifty-six centimeters, in length. Rotch states that there is very little variation in these measurements—not more, in his experience, than five inches, or 12.7 cm. It is generally taught that there is comparatively little increase in length in the first three or four months of life, but during this time the whole intestine grows more rapidly than does the sigmoid flexure, which at birth forms about one-half the entire length of the large intestine—indeed, it is stated that the sigmoid flexure actually diminishes in size owing to a readjustment of the mesentery (McClellan). Treves states that at the end of the first year the large intestine measures two feet, six inches, or 76 cm. in length; at six years about three feet, or 91.5 cm., while at thirteen years its length will be three feet, six inches, or 107 cm. The course of the colon is from the right iliac fossa upward to the liver, from which point the bowel passes transversely in somewhat of an arch across the abdomen to the spleen, forming, as it curves, the hepatic flexure, the splenic flexure, and the sigmoid flexure, the latter curve occurring in the left iliac fossa. Beyond this point it terminates in the rectum. Not infrequently its course may lie diagonally across the abdomen, from the hepatic region to the left groin. The sigmoid flexure is not only much more convoluted than in later life but sometimes it presents actual angulations. Undoubtedly the anatomy of the sigmoid flexure is closely related to certain pathologic states in infancy (see Constipation). In children the transverse colon, when distended with gas or fecal matter, can be quite clearly outlined by percussion.

The *cecum* occupies a higher position in the child than it does in the adult, and is also somewhat shorter in the former than in the latter. In thirty out of thirty-five cases examined by Rotch the position of the cecum varied from the right lumbar region to the lower part of the right iliac fossa. It is completely invested by peritoneum except on its posterior surface. As to whether or not the peritoneum covers the

latter site, authorities differ; thus, Treves, in his Hunterian Lectures, states that in 100 observations he always found the peritoneum infolding the cecum on its posterior surface, and Dwight (quoted by Rotch) found that out of thirty-seven young children the cecum was completely covered with peritoneum in thirty-three cases, and in the other four cases the largest part of the posterior surface of the cecum was invested by peritoneum. It is stated that during childhood the cecum is much more movable than in adult life.

The position of the *vermiform appendix* is usually behind the cecum, with its course directed upward and outward. Externally, it can be diagnosticated in case of operation by incising in the semilunar line upon the right side, at a point midway between the umbilicus and ileum (McClellan). In children it can sometimes be outlined by rectal or recto-abdominal palpation in cases where it is swollen. Great variation in the length and course of the vermiform appendix is frequently seen. It may be wholly or partially covered with peritoneum, the length of the attachments of which may vary considerably.

The *sigmoid flexure* consists of a good-sized loop of the large intestine, which occupies the left iliac fossa, although it is occasionally found in the pelvis; the mesenteric attachment here is relatively broad, allowing of quite a considerable amount of movement. The point at which the sigmoid flexure of the colon becomes the rectum is of interest from a surgical standpoint because it is at this point that stricture of the colon is most apt to occur. This is probably due to the arrangement of the bands of peritoneum, the so-called sigmoid mesocolon, which bind the loops of bowel so closely together that twisting on their axes may readily occur.

The lower portion of the large intestine, known as the rectum, is straighter in childhood than during adult life. This is due in part to the straightness of the sacrum at this time. (One sometimes notes persistence of the three rectal valves.) The peritoneum is reflected over the upper portion of the rectum in the same manner as in the adult, except that in the child it is lower down; the attachments between the rectum and the surrounding parts do not extend so high in children as in adults. The levator ani muscle does not perform its useful function so well in early childhood as in later life.

#### INTESTINAL DISCHARGES

Following birth, the first discharge from the intestines is of a dark-brown or greenish-brown color, which, from its resemblance to the inspissated juice of the poppy, is called meconium. This substance is

odorless, has a somewhat acid reaction, and is composed of partly digested amniotic fluid, cells, cholesterin crystals, and the constituents of bile. During infancy, while the child is fed on a milk diet, the intestinal discharges are of a light-yellow color and of liquid consistency, containing about 85 per cent. of water. They are feebly acid in reaction and contain fat, traces of bilirubin, mucus, intestinal epithelial cells, lime salts, and bacteria. In diseased conditions they may also contain false membrane, pus, blood, parasites and their ova, or even foreign bodies. The amount of intestinal discharge during the first few days of life will average from one to two ounces, and the number of stools varies from about two to five a day. As the child advances in age the number of bowel movements become gradually fewer, until the average attains about that of adult life—namely, one or two in twenty-four hours. In health the feces do not change in color until the child begins to take starchy food, when the brown color appears. The bacteria which are found in the intestinal discharges vary in number and species; many varieties have not yet been isolated; however, the *proteus vulgaris*, the *bacillus lactis aerogenes*, and Brieger's *bacillus* may any or all be found. When the infant begins to take a mixed diet, various forms of bacilli appear, among them the *streptococcus coli gracilis* and others. The color of the fecal discharges varies slightly in health, depending, to a certain extent, upon the character of the food. Certain medicinal agents, especially bismuth, iron and hematoxylin, will darken the color of the bowel movements. The color of the fecal discharge is changed greatly in disease; thus we have the characteristic clay-colored discharges seen in certain forms of intestinal disease or in pathologic conditions wherein the bile is not poured into the intestinal tract; the green-colored stool, found in the majority of forms of acute intestinal infections of moderate severity; and the peculiar watery discharge mixed with shreds of mucus—the “rice-water” passage of cholera infantum.

The bowel movements may be increased or diminished in disease. When abnormally large and frequent, they usually indicate a lack of capacity of the intestine to absorb nourishment; this is found in conditions of low vitality. When the lower bowel is chiefly affected and nutrition but slightly interfered with, the movements are frequent and small.

The characteristic odor of the feces is changed in disease; thus, when acid fermentation is present, they have a sour smell, or when decomposition of albuminoid matter has occurred, they are putrid or highly offensive. Constant feeding on broths will produce the same

result from the same cause. The musty odor of the stools of cholera infantum is well known.

Fragments of membrane or, rarely, casts of the intestine are sometimes found in the stools of children suffering from chronic (croupous) enteritis. Pus is seen in cases of severe ulceration of the lower bowel and in bad cases of chronic ileo-colitis. Blood in the stools may be the sign of the presence of an ulcer of the rectum or fissure of the anus, or rectal polyp; it is also seen in severe inflammation, especially of the lower bowel, and in intussusception. It is not infrequently seen in hemophilia. Dark or partly digested blood may be seen in the stools when the child has sucked from a fissured nipple; it is also a symptom of many other conditions, such as congenital malformation of the bile-ducts and diseased conditions of the liver.

Melena in the new-born is usually accompanied by hemorrhages from the bowels (see description of this disease).

## THE HEAD

The average circumference of the head at birth, the measurement being taken on a level with the middle of the forehead in front and the occipital protuberance behind, is about thirteen to fifteen inches, or thirty-four to thirty-seven centimeters. The longest diameter, taken between the middle of the lower border of the inferior maxilla to a point midway between the fontanels, measures at birth 13.5 centimeters or about 5 5/8 inches.

These measurements will, as has been before stated, bear a certain amount of relation to the measurement of the thorax and abdomen, and also to the entire length of the child at term. The shape and contour of the head and general topographic anatomy differ widely from that of the adult; thus we see that the cranial bones during early life are softer and capable of much greater compression than at a later period of development. Between the two divisions of the frontal bones at their upper angles in front and the superior and anterior angles of the parietal bones we find an opening caused by the lack of osseous deposit in the bones which form the boundaries of the space. This space is covered by skin and periosteum, and is known as the "anterior fontanel." The situation of this space usually corresponds with the junction of the sagittal and coronal sutures. Posteriorly, at the junction of the sagittal and lambdoidal sutures, is a smaller fontanel, known as the "posterior fontanel." The posterior fontanel usually closes soon after birth, the anterior one remaining open until

about the end of the second year, or, more accurately, at about the twentieth month. The size of the anterior fontanel at birth is about  $1\frac{3}{4}$  by  $1\frac{1}{4}$  inches (4 by 3 cm.). According to some authorities, the anterior fontanel increases in size from birth up to about the ninth month, after which it slowly grows smaller.<sup>1</sup> Occasionally supernumerary bones, called Wormian bones, which may vary considerably in number and size, are found in the sutures between the bones of the skull or sometimes within the fontanels. Their most frequent site is in the course of the lambdoid suture or in the posterior fontanel; they are rarely found in the anterior fontanel. The level of the scalp covering the fontanels is usually about the same or very slightly below that covering the skull generally; this, however, is greatly modified in disease. The pulsations of the cranial circulation can be distinctly felt at the anterior fontanel. The relation which the size of the face bears to that of the cranium is vastly different in the infant from that which we see in adult life. In the infant and during early life the face is much smaller in proportion to the size of the cranium than later. According to Froriep, the proportion between the size of the face and that of the cranium at birth is as 1 to 8, while in the adult it is as 1 to 2. While the height of the orbit bears nearly the same proportion to that of the skull during infancy that it does in adult life, the combined spaces of the two orbits equal nearly half the size of the face in infancy, while in the adult they equal slightly less than one-third. The lower border of the nasal opening during infancy is a little below the lowest point of the orbit, while in the adult it is very much below. The breadth of the skull, measured between the most distant parts of the zygomatic arches, bears a relation to the height of the face of about the proportion as 10 is to 4, this proportion being much smaller in the adult. In infancy the lower jaw is nearly on the same plane as the mastoid process of the temporal bone, and the upper border of the zygoma is on a level with the floor of the nasal cavity. In the adult the upper border of the zygoma is at or near the level of the floor of the orbit (Retch). The gums of the new-born infant do not meet. A lateral aspect of the skull of the new-born will show that the auditory meatus is situated at a point about the center of a line drawn along its inferior margin, while in the adult it is decidedly posterior to the center of this line. In infants and children the skin of the scalp is thicker than that of any other part of the body, and is closely adherent to the aponeurosis of the occipitofrontalis muscle. The pericranium

<sup>1</sup>We cannot subscribe to this view. An increase in the diameters of the fontanel should be viewed as an evidence of rickets as of increased intra-cranial pressure.

is but lightly attached to the bones of the skull, being intimately blended at the sutures with the membrane between the soft bones of the child's head; it is lax and admits of extravasations of blood beneath it, producing what is known as cephalhematoma.

The relation between the development of the head and thorax is exceedingly interesting. The circumference of the head, which is thirty-seven centimeters, or fifteen inches, exceeds that of the thorax, the latter being thirty-five centimeters, or fourteen inches. In the majority of cases this excess in the size of the head continues throughout the entire first year. During this time, however, the thorax increases in size at a more rapid rate proportionately than does the head, until, at the beginning of the second year, we find the circumference of the thorax slightly in excess of that of the head, and from this time on the thoracic circumference continues greater than the cranial.

TABLE SHOWING PROPORTIONATE SIZES OF THE HEAD AND THORAX FROM BIRTH TO THE THIRTEENTH YEAR—(Rotch)

(The Subjects of these Observations were Male Children)

Age	Head	Thorax
At birth.....	37 cm. (15 inches).	35 cm. (14 inches).
2 years.....	48 cm. (19 inches).	51 cm. (20 $\frac{1}{8}$ inches).
3 years.....	51 cm. (20 $\frac{1}{2}$ inches).	55 cm. (21 $\frac{3}{4}$ inches).
4 years.....	53 cm. (21 inches).	54 cm. (21 $\frac{1}{2}$ inches).
5 years.....	53 cm. (21 inches).	54 cm. (21 $\frac{3}{4}$ inches).
6 years.....	52 cm. (20 $\frac{1}{2}$ inches).	55 cm. (21 $\frac{3}{4}$ inches).
7 years.....	54 cm. (21 $\frac{1}{2}$ inches).	54 cm. (21 $\frac{1}{2}$ inches).
8 years.....	53 cm. (21 inches).	59 cm. (23 $\frac{1}{8}$ inches).
9 years.....	54 cm. (21 $\frac{1}{2}$ inches).	61 cm. (24 inches).
10 years.....	53 cm. (21 inches).	62 cm. (24 $\frac{1}{2}$ inches).
11 years.....	56 cm. (22 $\frac{1}{8}$ inches).	63 cm. (24 $\frac{3}{4}$ inches).
12 years.....	53.5 cm. (21 $\frac{1}{8}$ inches).	63 cm. (24 $\frac{3}{4}$ inches).
13 years.....	54 cm. (21 $\frac{1}{2}$ inches).	66 cm. (26 inches).

## BRAIN

At birth the dura mater is closely adherent to the skull; in fact, so intimately are they connected that extravasations cannot take place between them. In the subarachnoid space a larger amount of fluid is found during infancy and throughout childhood than in adult life; the quantity of fluid present is generally just about sufficient to fill the space comfortably. McClellan (quoting from Hilton) states that

hydrocephalus, due to an excessive amount of fluid in the ventricles of the brain, may be caused by a closure of a small opening in the pia mater, which is found at the inferior boundary of the fourth ventricle, and which is known as the foramen Magendie. The blood-vessels of the pia mater are so exceedingly delicate that high blood pressure, traumatism, etc., may readily cause hemorrhage into the subarachnoid space; and from this cause monoplegia, hemiplegia, or diplegia may result. During fetal life and, indeed, from birth up to the seventh year the growth and development of the brain are very rapid, but after the seventh year the growth, although steady, is slow. Cellular multiplication in the cortex of the brain ceases at the third month of fetal life; though many of these cells are in an immature state until a much later period. At birth the weight of the brain is one-third that of the adult encephalon. At the seventh or eighth year the adult size and weight are practically attained, though there may be a slight increase in both up to the twenty-fifth year. This rapid attainment of weight is due to the relatively greater amount of medullary matter in the child's brain; subsequent growth is represented by an increase in the thickness of the cortex and in the size of the cortical constituents, as has been pointed out by Boyd, Veirordt, and Bischoff. The association pathways of the brain are the last structures to be developed, and are probably not completely formed to the pubertal age or beyond it. At birth the brains of male and female children are practically the same size, but subsequently the brain of the male grows more rapidly than does that of the female.

One of the most important points of external difference between the brains of the child and the adult is that the fissure of Sylvius, in its relation to the sphenoparietal and squamous sutures, occupies a higher position in childhood than in later life. Both Symington and McClellan found, in a large number of examinations and dissections of frozen sections of the brains of children of various ages under seven years, that the Sylvian fissure was always above the squamous suture and was covered by the parietal bone. The position of the fissure of Rolando is about the same in the child as in the adult. According to Huschker, the cerebellum is much smaller, as compared to the cerebrum, at birth than later in life. The convolutions of the brain in the infant are slightly more shallow and have a much less complex arrangement than in the adult. The depressions or sulci between the convolutions are not so deep in early as in later life. The highly specialized centers of the brain are apparently not fully developed in the young infant.

## SIGHT

From an anatomic standpoint, the eye is fully developed at birth and is sensitive to light; but the visual pathways are not developed. There is no capacity, therefore, to interpret the images received. Apparently, the infant can distinguish light from darkness at a very early age, and shows pleasure in looking at a light or a bright object. About the third or fourth month a baby will usually begin to know its mother or nurse, and by the sixth month many objects with which the child is in constant association can be recognized. The power to estimate distance and to coordinate movements by sight is not developed until some time later. It is said that an infant cannot appreciate color until after one year of age, and then only the brighter hues. Yellow and red are the earliest colors perceived. According to Preyer, the movements of the eyes are not coordinated during early infancy, and perfect consensual motion is often not attained until the fourth year.

## HEARING

Apparently the sensation of hearing is not fully developed at birth. It has been stated that this is possibly due to the absence of air from the tympanum, to the presence of fluid in the middle ear, and to a swollen condition of its mucous membrane. An infant is able to hear a loud sound by the third day of life. Failure to hear sounds as late as the fourth or fifth week may show that the child is deaf or idiotic. The power to appreciate the direction of familiar sounds appears about the third month, and from then on the development of the hearing mechanism proceeds more rapidly than does that of the visual. The sense of *touch*, *taste*, and *smell*, are probably quite well developed at birth. *Sensibility* to touch, temperature, and pain is probably not very acute in early infancy.

## LACRIMAL GLANDS

At birth the lacrimal glands are not fully developed. It is rarely that a baby sheds tears before the third or fourth month. When an infant is seriously ill no tears are shed, and their appearance is said to be a sign of beginning improvement.

## SALIVARY GLANDS

The secretion of saliva is not fully established in the new-born infant, and in consequence of this we find the mucous membrane of

the mouth quite dry. The starch-digesting function of the saliva is very slightly present, if present at all, at birth.

The teeth will be considered in another section (Gastro-intestinal Diseases).

### THE SWEAT-GLANDS

Young infants rarely perspire to any marked degree except when overheated, although the skin may be very slightly moist, especially during sleep. Profuse and continued perspiration is usually a symptom of rickets, and this is particularly the case when the sweating is greatest about the head and increases during the night.

### SEBACEOUS GLANDS

As evidence that these glands are capable of performing their function before birth we have the vernix caseosa covering the body of the new-born child. The scalp of the infant may later become covered with a yellowish, scaly secretion known as seborrhœa capitis.

### THE TESTICLES

Under normal conditions the testicles descend through the inguinal canal into the scrotum during the ninth month of intra-uterine life. It is not uncommon, however, to see children in whom one or both testicles are still in the abdominal cavity, and some of these patients will need surgical care. If the descent of the testicles is greatly delayed, a portion of intestine may follow its descent down through the canal into the scrotum. It is probable that these glands furnish an internal secretion of value.

### THE BREASTS

The breasts of infants of both sexes may be somewhat enlarged at birth, and may contain a milky secretion which, under the microscope, resembles colostrum. Should this secretion become so increased in quantity as to cause great distention of the breasts and pain, the milk may be pressed out by gentle massage and a small breast-binder applied; but any manipulations must be most gentle. Usually it is better to protect these swollen breasts and let them alone. Great care should be exercised to keep the nipples aseptically clean or an abscess may follow. The condition usually disappears toward the end of the first month.

## THE EXAMINATION OF THE SICK INFANT OR CHILD

How shall one approach the sick child? Practically, as others have stated, the examiner may adopt one of three attitudes toward the little patient: The first is one of frankness and good fellowship, and it may be assumed with the normal, well-trained child. Secondly, with the neurotic or severely ill child he must exercise tact. And lastly, with the spoiled prodigy, openly resisting his efforts to examine, he must resort to that which is most expeditious as well as most kind—absolute coercion (make him do exactly what you wish him to do). When the child is very nervous or quite ill, it may be well to adopt Louis Starr's suggestion and secure the history in an adjoining apartment.

*The History.*—History taking, so often considered mere clerical work, soon becomes one of the pleasures of medical life, and while doing a greater justice to the patient, it never fails to broaden the mental horizon of the examiner.

After securing the name and age of the patient, one should ask some questions concerning the school life—that is, if he is old enough to attend school. One so gains a general insight into the mental and social condition of the patient.

“How long is it since the child was perfectly well?” That canny question of Professor Tyson well initiates our history. If one simply asks “How long has he been sick?” one may elicit only the history of a recent acute illness.

The next question asked is: “Why did you bring the baby to see me?” or “Tell me just why you sent for me.” This enables the mother to describe the illness in her own words.

One needs no card with a set form for the history—the plan should exist in one's mind. The history is best written on a blank card, small enough to be carried in the pocket and yet large enough to contain a respectable account of the illness, etc.

The convenient method to pursue from now on is a chronological one. From the ideal standpoint, one should always elicit a family history, for no knowledge can be valueless, and yet to the busy practitioner such thorough work is not always feasible. The questions asked above are important sign-boards and their answers may make such a complete history imperative. To illustrate: Measles, in a previously healthy child may require only the history of exposure, onset and prodromes, but in another case there may exist a tuberculous taint, and the history may prepare us to forebode the little patient.

The family history naturally divides itself into three parts: First, the parental history; second, the investigation of the grandparental history and that of collateral branches of the family; third, the inquiry concerning other children in the family.

1. *Maternal and Paternal History:*

- A. *Search for syphilis*, being careful not to arouse unjust suspicions, and not neglecting to ask the occupation of the father.
- B. *Tuberculosis*, not so much from the standpoint of heredity, as to elicit the possibilities of tuberculous environment.
- C. *Insanity or mental deficiency*.
- D. *Organic nervous diseases or neuroses*.
- E. *Alcoholism*.
- F. *Gout*.
- G. *Rheumatism*—not alone of the acute articular variety, but including Sydenham's chorea, tonsillitis, erythema-multiforme, peliosis-rheumatica, etc.
- H. *The health of the mother during pregnancy*.

2. *Grandparental and Collateral History:*

This is of importance when we are dealing with:

- A. *Hemophilia*.
- B. *Nervous affections*, particularly of the family type or with epilepsy.
- C. *Psychoses*.
- D. *Gout*.

3. *The Inquiry Concerning the Other Children:*

- A. *The number of pregnancies*.
- B. *Abortions, miscarriages, etc.*
- C. *Still births*.
- D. *The number of children living and dead*.
- E. *The cause of the deaths*.
- F. *The health of the living children*.

*The Personal History.*—We still pursue the chronological order, beginning at birth. Was the patient a full term baby or was he premature? Was the labor difficult, unduly prolonged, precipitate, or instrumental? Right here it is important to remember that difficult or prolonged labor may jeopardize the life and health of the child far more than skilfully employed instruments. Was there cyanosis at birth, or soon after and of what degree? How soon did the new-born

infant cry? What was the birth weight and state of nutrition? Have snuffles or early skin eruptions (roseola, pemphigus, etc.) been noted in early life? Sometimes when this question is answered in the negative, it is wise to inquire about "baby measles."

Has the mother been able to nurse the baby? If not, what other foods have been given? In either case, has the baby been fed at regular intervals? In difficult feeding cases these questions must be elaborated and every phase of the feeding question gone into to its most minute detail. Inquire in the case of breast-fed babies concerning the time of weaning, what foods were used and with what results. Have the babies been habitually constipated or the reverse? Has there been vomiting, and if so did it occur soon after eating or after a lapse of time? Has he advanced properly in weight? Then turn to the other important phase of infantile existence, sleep, and ask concerning the amount of sleep, etc., its regularity, depth and disturbances.

Next we inquire about dentition. When was it initiated? Were there teething disturbances? So we may find important evidence bearing upon rickets, cretinism, etc. Next investigate the motor development during the period of infancy. Delay in such development may often be fraught with significance. The acquirement of speech probably furnishes us with the best evidence of the baby's mental development. Nor should one forget in certain cases, the sensory side of the nervous system. Make inquiries regarding the development of the special senses. What diseases did the baby have during the first and second year? Special attention should here be paid to infections of the new-born, the diarrheas of the summer season, convulsive disorders and catarrhal pneumonia.

Then we investigate the question of the exanthemata and the other infectious diseases of childhood, and anent of one of them, inquire about successful vaccinations. Nor do we forget in this inquiry to include acute articular rheumatism, and the other rheumatic conditions before mentioned. In the presence of an existing exanthem we pursue a special order of questions: 1. What has he had and has he been vaccinated? 2. To what has he been exposed and when was he exposed? 3. What prodromes has he exhibited?

From now on we desert the chronological order and turn to the various systems and organs of the body, asking questions about gastrointestinal, respiratory, urinary, nervous and other disturbances, much as is done in securing the history of the adult. Remember though, that the school life and the pubertal period may be productive of more or less peculiar disorders.

It is probably unnecessary to say that so formidable a list of questions is not asked, and cannot be asked of every parent whose sick child needs medical attention. It is our purpose to point out a useful order that may be followed in the elicitation of the history. But, on the other hand, we must hasten to add that it is sometimes necessary to ask more questions than these; we must also say that one never takes a thorough history in which some important fact is not elicited that would otherwise have remained undetermined.

The objective or physical examination of the patient: Certain general observations are made almost intuitively by the skilled observer. Thus he can scarcely avoid thinking at first glance that a certain child is not seriously ill; he is equally certain that another is well-nigh moribund; that still a third is profoundly septic. So he promptly attributes to one child a sturdy constitution and immediately dubs another delicate. Peculiar postures, often so significant; abnormalities of movement possibly almost pathognomic; certain skin eruptions; jaundice; the color of the skin in cardiac diseases, cachexias, etc.; labored or panting respirations, etc., are all phenomena that attract the observant clinician's attention immediately.

Some of the great clinicians of olden times were wonderfully keen observers, but no acuteness in general observation could or ever can equal the modern diagnostic measures of systematic examination, and the employment of instruments of precision. A certain medical student suffered from severe pain in his left side. He consulted a brilliant professor, who on two occasions, told the young man that he was smoking too many cigarettes, and advised him to desist. The habit was accordingly stopped, but the pain continued. The student then consulted a phlegmatic and painstaking subordinate to the great professor, and this man quickly found a massive pleural effusion. The noted professor detected the stained fingers of the cigarette smoker, and jumped to a conclusion; the man of slower mind examined his patient and justified his method. So let no general observations overshadow or supplant the results of systematic physical examinations.

Goodhart gave us a good rule to follow in the examination of a sick child, *viz.*, in cases of doubt examine the mouth and throat; examine the chest; examine the abdomen. At present we have a better rule—examine the sick child from the crown of his head to the soles of his feet, accepting in addition to this any aids the laboratory may furnish.

Let us proceed to make such an examination, apologizing in ad-

vance for what must be a mere enumeration of some important points to be noted.

1. *The hair and scalp*, one may observe:

- A. The coarse dark hair of the Mongolian imbecile.
- B. The coarse dry, scanty growth of the Cretin.
- C. The bald spot of rickets.
- D. The various parasitic scalp diseases.
- E. Head sweating of rickets.
- F. The denuded areas of alopecia.
- G. Ulcers, or scars from syphilis.

2. *The Skull*:

- A. The square, the asymmetric and other types of rachitic skulls.
- B. The large and globular skull of hydrocephalus (McEwen's sign being sometimes obtained over the pterion).
- C. Delayed closure of the fontanelle in rickets. The bulging fontanelle of meningitis or hydrocephalus. The depressed fontanelle in atrophy or where much fluid has been lost by the economy.
- D. The small and peculiarly shaped skull of microcephalus.
- E. Meningocele, encephalocele and meningoencephaloceles.
- F. The cranio tabes of rickets and syphilis.
- G. Bony losses from lues.
- H. Fractures, depressions, clefts, and exostoses.

3. *The Face*:

- A. The complexion and various discolorations of the skin.
- B. The pasty complexion and edematous eye-lids of the Cretin.
- C. The characteristic Mongolian features.
- D. The expressions of mental deficiency and of the rarer insanity, of the totally blind or deaf, etc.
- E. The furrowing of the brow with headache or eye strain, the nasolabial lines and playing *alæ nasi* with lung affections, and the pronounced oral lines of abdominal disease.
- F. The large bones of acromegaly.

4. *The Eye*:

- A. The Mongolian slant of the eyes.
- B. Exophthalmos.
- C. Squint.
- D. Conjunctivitis or keratitis.

- E. Corneal ulcers or leukomata.
- F. Iritis, from syphilis, etc.
- G. Inequalities or irregularities of the pupil, so important in the diagnosis of meningitis.
- H. Congenital cataracts or anomalies of the lens.
- I. The manner in which the child examines pictures or print.

All of these may be observed by the careful clinician, while the expert ophthalmoscopic examination may determine much more: for instances evidences of brain tumor, tubercle, amaurotic-family-idiocy, etc.

5. *The Nose:*

- A. Its broadened bridge and poor streams of air may direct attention to adenoids (a wisp of cotton held in the stream will very readily detect the occlusion of one or other of the nares).
- B. Its broken down bridge may furnish indubitable evidence of hereditary syphilis.
- C. The hemorrhage from one nostril may indicate a foreign body, nasal diphtheria or syphilis.
- D. Bilateral hemorrhage may point toward adenoids or may have much the same significance of constitutional or local disease that it possesses in later life. Though one does not essay to be a specialist, one should be able to use a nasal speculum.

6. *The Mouth and Throat:* Observe.

- A. The rhagades of syphilis.
- B. The herpes-labialis that may possess so much diagnostic import.
- C. The oral breathing, that in waking or sleeping hours, should make us think first of adenoids.
- D. The crowded and distorted teeth that oblige us to consider the same condition.
- E. The characteristic teeth of syphilis (these being seen only in the second dentition).
- F. The dental caries that may represent the portal of entry of the tubercle bacillus, and explain the subsequent involvement of the cervical glands.
- G. The tongue may reveal macroglossia, the characteristic appearances of scarlet fever, and many other features of importance. The rare ranula may appear under it.

- H. The cleft or asymmetric palate should not escape detection.
- I. Acute inflammations of the mouth and throat are, of course, noted (the Freeman method of examination being usually employed).
- J. The Koplik's spots of measles.
- K. The chronically enlarged tonsils, particularly of the irregular variety, may accompany adenoids; but never rest satisfied with a mere inspection of the nasopharynx if certain conditions are suspected; for
- L. The digital examination may reveal not only adenoids, but also postpharyngeal abscesses or foreign bodies. (One of us so detected a jack stone only a few weeks ago.)

7. *The Ear:*

- A. Tenderness over the tragus or mastoid processes may suggest a proper diagnosis.
- B. We cannot all become skilled otologists, but we may and should learn to use the aural speculum so well that impacted cerumen or a bulging lusterless ear drum may be seen. Many an obscure diagnosis is so made clear.

8. *The Neck:*

- A. The presence or absence of thyroid gland (if the gland is present the isthmus may always be felt).
- B. Goiters.
- C. Congenital fissures, or branchial cysts.
- D. The undue pulsation of the arteries, the venous pulse or possible diastolic collapse of the cervical veins (Friedreich's sign).
- E. The various cervical chains of lymph nodes: submaxillary, submental, tonsillar, deep or jugular, superficial and supraclavicular.

9. Indeed, at this point it is well to study the other accessible lymph nodes of the body; otherwise, we find we are liable to forget them. These are the axillary, epitrochlear, inguinal, femoral and the popliteal. It is scarcely necessary to mention that adenopathies (general or local) may be indicative of syphilis, tuberculosis, Hodgkin's disease or certain other infections. In the presence of a localized glandular enlargement, one should always think of the areas its lymph vessels drain.

10. *The Chest:*

The scope of this chapter will permit but a cursory mention of important thoracic and intrathoracic affections: Inspect, palpate,

percuss, combine auscultation with percussion, measure when necessary; and when these preliminary methods have been pursued, not until then, listen to the cardiac and breath sounds. In ausculting, too, a definite order may be pursued with advantage: *viz.*, study the first sound of the heart, the second sounds, the adventitious sounds; then the inspiration, the expiration, the adventitious sounds accompanying one or the other, or both.

- A. Malformations of the chest may mean much to the examiner and patient. Thus, the violin-shaped chest of rickets; the funnel-shaped chest (trichter-brust) of adenoids, furnish striking evidences of their respective causes.
- B. Nor should we forget the rickety rosary, one of the earliest evidences of rickets.
- C. Congenital cardiac diseases, or vascular anomalies may be associated with or indeed the causes of marked bodily dwarfing and thoracic malformations.
- D. The most common pleural effusions in early life are empyemata; although interlobar empyemata may occur.
- E. Remember the significance of manubrial dulness and stripe dulness (combined they point toward enlargement of the mediastinal glands, most often toward tuberculosis of them). The mention of these phenomena reminds us that percussion in the infant or child is a distinct art, different from that pursued in the examination of the adult's chest. (Dr. Samuel McC. Hamill has recently contributed an important paper upon this subject.) Three aphorisms will serve us well: First, percuss lightly with one finger, not with a percussion hammer; second, percuss for your own information, not for demonstration to others; third, study not only the percussion note but also the resistance offered to the finger or pleximeter.

## II. The Abdomen:

Here the same diagnostic procedures stand us in good stead; though abdominal diagnoses may be most difficult. Indeed, pre-operative diagnoses may prove impossible. The writer firmly believes, however, that such difficulties are minimized in childhood.

Auscultation, it is true, ceases to be a court of last resort; though combined with percussion it may still yield evidence of much value. Palpation, on the other hand, looms prominent, for one may truly acquire the *tactus eruditus*. (It is our custom to follow Edebohls'

method of palpating the abdomen, namely, to place the hand perfectly flat upon the abdomen, thus distributing the pressure; to approach the organ to be palpated; and in superficial palpation to flex only the terminal phalanges.)

Some of the important things to be noted—we can mention but a few—are:

- A. The pot belly of rickets, Cretinism or Mongolianism.
- B. The retracted, or scaphoid abdomen of meningitis.
- C. The various forms of hernia and diastasis of the recti muscles.
- D. The pouting umbilicus, enlarged superficial veins and protruding abdomen of tubercular peritonitis.
- E. Enlargement of the liver or spleen, easily detectable by the Edebohl method of palpation, and also by such light percussion as has been described.  
Such enlargements may occur in syphilis, rickets, malaria, certain anemias, infections, and in other conditions whose causes and nature are not determined.
- F. The tenderness and increased muscular resistance in appendicitis—remembering that both may be much less pronounced than in adult appendicitis. In early life, too, the appendix may occupy a considerably higher position than it does in later years.
- G. Abdominal tumors, particularly malignant growths of the kidney, and the “sausage-shaped” tumor of intussusception.
- H. Distention of the urinary bladder.
- I. Distention of the hollow viscera, or sagging downward of the same.

## 12. *The Back:*

No examination of the trunk is complete until one has examined the spine and great muscular masses of the back: Spina bifida (of several varieties), lateral curvature, Potts' disease in an incipient or advanced form, lordosis of the lumbar region in rickets, Cretinism, etc., are but a few of the important conditions to be observed.

13. Rectal examinations under anesthesia are occasionally demanded and the younger the infant, or child, the more accessible to the finger are certain pelvic, abdominal and retroperitoneal pathologic conditions.

14. *The Genitalia:*

- A. Many anomalies may be seen, though we shall refrain from their enumeration.
- B. One must mention the frequency with which preputial adhesions occur—not forgetting that the same conditions may obtain about that feminine analogue—the clitoris.
- C. Nor must one fail to note the vaginal discharges of little girls.

15. *The Limbs:*

- A. Anomalies and malformations of the limbs.
- B. The extremities of dwarfism, not failing to observe that peculiar dwarfing of achondroplasia fetalis.
- C. Giantism, particularly in connection with the disease acromegaly.
- D. The distorted limbs of rickets.
- E. Rheumatism, recalling that its articular manifestations in children may be very slight.
- F. Syphilis of bones, periosteum, epiphyses, or joints.
- G. Tuberculosis of joints.
- H. The subperiosteal hemorrhages of Barlow's disease (infantile scurvy).
- I. The stubby fingers of the Cretin; the trident hand of achondroplasia; or the bowed little finger of the Mongolian.
- J. The enlarged or wasted muscles in the muscular dystrophies.

16. *The Skin:*

Now that the patient has been thoroughly undressed, make a careful examination of the skin surface, searching for scars, exanthemata, birth-marks, etc.

17. Where two or three photographs of the patient can be taken they sometimes furnish a graphic record worth more than many pages of description.

18. The physical examination should be completed and supplemented by a study of the urine and blood; or if the need exists, by a study of the sputum, of the stools, by a culture from the throat, or by an exhaustive examination of the cerebrospinal fluid. Nor must X-ray diagnosis be excluded from this laboratory category. Yes, to be just, we must mention opsonic work too.

Every student should read a recent address of Dr. Herrick's, "The Relation of the Clinical Laboratory to the Practice of Medicine." Laboratory methods cannot supplant the older diagnostic procedures; but like other diagnostic measures, they can and should play their parts. Very wonderful rôles they are too; for not seldom they make the diagnosis when the older measures fail. Thus a negro boy, five years old, studied at the Polyclinic Hospital had inequality of the pupils, retraction of the head, a right hemiplegia, aphasia, convulsions, etc. His temperature was often subnormal. The hematologic study showed the presence of the Widal reaction and of leukopenia, while the examination of the cerebrospinal fluid proved to us that he had a typhoidal infection of his meninges.

Though not laboratory men, we say with Herrick: "Let us have more laboratories, not less; more for the practitioner and more for the students." Encourage the young men in our midst to do the laboratory work that we cannot do. Let us read Margaret Deland's essay upon the "Sinfulness of Growing Old," and if we find ourselves prejudiced against these newer methods, let us arise and smite the invader. If we can save ourselves from prejudice then shall we remain young to the last.

Having pursued the physical study thus thoroughly, how intelligently one may enter upon the nervous examination. Possibly the cause of the nervous or mental disease is already clear and it simply remains for us to localize the lesion. The scheme of nervous examination followed by the writers is practically that of Dr. Wm. G. Spiller.

<i>Motor</i>	{ The paralysis or paresis (hemiplegia, diplegia, monoplegia, etc.). Muscular wasting; loss of sphincter control; station and gait; the reflexes; electric responses; phenomena of irritation.
<i>Sensory</i>	{ Tactile, temperature and muscular sense; vision; hearing; taste; smell.
<i>Mental</i>	{ Feelings; emotions; moods; memory; impulses, etc. Aphasia should receive much study.
<i>Vaso-motor and trophic</i>	{ Flushing; pallor; cyanosis; tache cérébrale. Trophic changes in the skin and its appendages and in the bones.

Let us close with a brief summary:

First. Know children.

Second. Learn the joys of history taking, if perchance you are not

acquainted with them. In securing the history follow a chronological order.

Third. Examine children thoroughly, placing the objective far above what we are told; and, above all, follow a method in such examinations.

Fourth. Aid and abet your diagnostic procedures with laboratory methods whenever such aids are needed and procurable.

## CHAPTER II

### DISEASES OCCURRING AT OR NEAR BIRTH

#### GROSS ANOMALIES

A number of these anomalies may be observed in new-born children, the accompanying illustration representing a remarkable example; but for a discussion of these we must refer the student to the classic work of "Hirst and Piersol," or "Gould's and Pyle's Anomalies and Curiosities of Medicine, etc.

#### ASPHYXIA NEONATORUM

**Synonyms.**—APPARENT DEATH OF THE NEW-BORN; ASPHYXIE DES NOUVEAU NES; ASPHYXIA PALLIDA NEUROSA

**Definition.**—Deficient oxygenation of the fetal blood.

**Etiology.**—Asphyxia in the new-born may be divided into (1) antepartum, or intra-uterine, asphyxia and (2) postpartum, or asphyxia occurring immediately after birth. The *causes of antepartum* asphyxia are: Partial or complete detachment of the placenta; interference with placental circulation, such as would be caused by pressure on the umbilical cord, or by the cord being drawn tightly about the child's body or neck; considerable nervous depression in the mother; loss of blood; continued depression of the fetal skull by the maternal parts; premature attempt at respiration by the fetus and consequent inspiration of the amniotic fluid or the secretions of the birth canal. If attempts at respiration during the child's passage along the birth canal are vigorous and prolonged, there may result a form of catarrhal pneumonia, known as "inspiration pneumonia," which may come on a few hours after birth and will probably prove fatal.

**Postpartum and Extra-uterine Causes.**—The most common cause of asphyxia following birth is imperfect development of the child. This may arise from the fact that the fetus is immature, the respiratory function on this account being so feeble that it cannot freely inspire air in sufficient amount to inflate the lungs, thus leaving areas of pulmonary vesicles in an unexpanded condition.

This condition of deficient pulmonary expansion is known as

atelectasis. The bony walls of the thorax may be too soft to allow of expansion through muscular action. Simple weakness alone in the child may be a cause of asphyxia following birth.

Disease affecting, by mechanical pressure, the respiratory apparatus, or structural changes in the latter, may be causes. We may also see asphyxia arising from syphilitic diseases of the liver or lungs or from pressure produced by dropsy or tumors.

Asphyxia following prolonged birth pressure may be caused by hemorrhage into the fourth ventricle or into the substance of the medulla, thus producing pressure on the respiratory centers. In other cases we may see hemorrhages into the lungs themselves, producing postpartum asphyxia.

Asphyxia of the new-born may be divided into three grades:

1. A slight suspension of respiration, due to an accumulation of mucus or a foreign body in any part of the respiratory tract.

2. There may be observed a type of asphyxia known as the livid, sthenic, or apoplectic form, seen in robust and full-blooded infants.

3. A condition in which the child is pale, limp, and apparently lifeless, and which is known as pallid, asthenic, or anemic asphyxia.

**Pathology.**—Postmortem examination will reveal patches of extravasation and ecchymoses in various organs, particularly the brain, meninges, and liver. The lungs are dark in color, firmer than normal, and engorged with blood. The air-passages are more or less filled with mucus, amniotic fluid, and meconium where attempts at intra-uterine inspiration have been made.

The cerebral sinuses are engorged with blood, and there is some edema of the membranes covering the brain.

In extra-uterine asphyxia the markings or alterations in the shape of the head will often be seen when pressure has been the cause.

In the lungs, on postmortem examination, areas of unexpanded



FIG. 2.—A STILL-BORN CHILD IN THE PRACTICE OF DR. WENDY. Specimen sent to the Samaritan Hospital by Dr. Samuel Wolfe. There were noted: Absence of both lower extremities (amelia); arrest of development of the left arm above the elbow; ankylosis of the right elbow-joint and complete atresia ani. The mother of this monster had performed numerous abortions upon herself. Her cervix was said to be exceedingly firm from cicatricial tissue. Possibly there were intra-uterine bands.

vesicles will frequently be found. The veins and the right division of the heart are distended. In the head, overlapping of the bones at the sutures, congestion of the cerebral sinuses and meninges, or hemorrhages into the latter, may be present.

**Symptoms.**—The symptoms of intra-uterine asphyxia can only be determined by close observation of the child while in the womb. On auscultation a very slow or very rapid fetal heart rate will indicate either pneumogastric irritation or paralysis.

As the asphyxia continues muscular spasms and unusual movements of the child may be observed. A practical point to be gained from the symptoms is that the movements and heart-beat of the child should be carefully examined before deciding on operative or other methods of delivery. When the umbilical cord is tightly wound about the child's neck or arm, a bruit, synchronous or slightly slower than the fetal pulse, can be heard by auscultation over the mother's abdomen.

Antepartum asphyxia may be suspected whenever a fetal heart-beat previously regular becomes either very rapid or very slow and faint, or when by vaginal examination, the pulsations of the umbilical cord weaken or cease, or meconium is passed by the fetus.

The symptoms of postpartum asphyxia vary considerably in the sthenic and asthenic forms. In the *sthenic*, or livid, variety the cutaneous surfaces are cyanotic, the face is dusky or blue in color, the muscles retain their tone, and may be somewhat rigid. Cutaneous irritability is present, and the eyeballs are prominent, with the conjunctivæ injected.

The pulsations of the cord are strong and full, the respirations intermittent, and reflexes may be excited by irritation. In the *asthenic* or pallid form the body is pale and limp and the extremities are quite cool. The face is white and death-like, and the lips are blue. The muscular system is relaxed, and there is little cutaneous irritability. The heart-sounds are slow, irregular, and often so feeble that it is difficult to tell whether pulsation exists or not. Relaxation of the anal sphincter is common. The cord is often thin and pale, while its vessels are nearly empty. This sort of asphyxia is often seen in infants of deficient general development. Not infrequently such a child may make a few irregular spasmodic attempts at respiration, the latter soon ceasing, the heart-beats becoming slower and feebler, and death results, or the child may survive a few hours and then expire.

**Prognosis.**—The prognosis will depend greatly on the promptness and efficiency of the treatment. Asphyxia in the new-born rarely

tends to spontaneous recovery. As a rule, the prognosis in the asthenic form is the more unfavorable. If auscultation for five minutes fails to reveal any heart-beats, the case is hopeless; otherwise efforts at resuscitation should be continued as long as any action of the heart can be detected. An unfavorable symptom is the continued weakening of the heart and lowering of temperature notwithstanding all treatment. It must be borne in mind in the prognosis that the dangers of asphyxia are not over with the immediate preservation of life, as the child may later perish from atelectasis, inspiration pneumonia, or from the effects of cerebral compression or hemorrhage.

**Treatment.**—The treatment of asphyxia in the new-born is divided into prophylactic and curative.

The *prophylactic treatment* consists of avoiding, so far as possible, impediments to the fetal circulation by the correction of faulty presentations, the judicious use of forceps, or, when the child cannot be saved by these means, the decision upon suitable operative procedures for its delivery.

*Curative Treatment.*—When a moderate degree of asphyxia is present, the feet of the child should be grasped firmly and in such a manner as to prevent its slipping from the physician's hands, and the head be allowed to hang downward, in order that the blood may gravitate toward the brain. While the infant is in this position the nurse should quickly mop the mouth and upper part of the throat with a piece of gauze or absorbent cotton wet with boric acid solution, in order to remove any mucus which may obstruct the air-passages. She must be very careful that no infection is carried to the child in this procedure, as infection of the mouth and throat by too severe and injudicious rubbing is by no means as uncommon as we might believe. One of the authors has seen two cases of general septic infection in two years;<sup>1</sup> in both instances the site of primary infection was the mouth and was caused in the way mentioned above.

The next procedure consists of placing the child in a hot bath at a temperature of about 100° F. (37.8° C.), and pouring or dashing a thin stream of cold water upon the chest or back; slapping the buttocks with a towel wet with cold water is often useful.

If these methods are not successful, the mucus or fluids obstructing the air-passages should be removed by suction through a soft india-rubber catheter passed into the trachea. Irritation applied to the skin, or blowing air over the child by means of a bellows or fan, occasionally does good.

If the child still fails to respond, a soft catheter should be passed

into the larynx and the lungs inflated, or the same result may be brought about by Richardson's bellows.

When a greater degree of asphyxia exists, any one or all of the following manipulations should be used successively:

*Schultze's Method* (see Figs. 3 and 4).—The physician, standing with the body slightly bent forward and the legs moderately sepa-



FIG. 3.—SCHULTZE'S METHOD (INSPIRATION).

rated, the arms extending toward the ground, seizes the infant by the axillæ in such a manner that his index-fingers are passed from behind forward, the back of the infant being toward the operator. The thumbs rest gently over the clavicles, against the posterior surfaces of which the remaining fingers are applied in a direction from above downward.

The infant's head is supported against the operator's wrists, who, while thus holding it, quickly raises the infant forward and upward until the operator's arms are somewhat above the horizontal line; the

infant's body is now in a state of extreme extension, and inspiration is produced thereby.

At the moment when the operator's arms are somewhat above the horizontal the motion is suddenly stopped, thus allowing the infant's body to flex upon itself in front of the operator's face. This movement



FIG. 4.—SCHULTZE'S METHOD (EXPIRATION).

flexes the infant's spine and compresses the thorax and abdomen, thus producing expiration.

The infant is now returned to the first position, and the manipulations are repeated at about the rate of from eighteen to twenty times a minute.

*Sylvester's Method.*—Place the infant on its back, with a small rolled towel between the shoulders, so as to extend the abdomen and thorax. Now grasp the arms above the elbows and bring them quickly upward by the side of the head, at the same time everting them. Next bring them down again against the sides of the chest, and make gentle

but firm pressure. The movements should be repeated at intervals corresponding with those of normal respiration.

*Laborde's Method.*—This consists of laying the child on a flat surface, with a small rolled towel between its shoulders. It is generally recom-



FIG. 5.—SYLVESTER'S METHOD (EXPIRATION)

mended that the head and neck be in the state of considerable extension. The tongue is seized with a pair of hemostatic or other small forceps, and drawn out and in with a rhythmic motion, the frequency of which should correspond with those of normal respirations. As the tongue



FIG. 6.—SYLVESTER'S METHOD (INSPIRATION).

is withdrawn, the operator counts *one*, as he relaxes the tongue, he counts *two*, and at the same time, he compresses the baby's chest with the other hand. It is recommended that this traction of the tongue be supplemented by motions of the arms, as in Sylvester's method.

A modification of the so-called Byrd-Dew method will often be

found very successful and is to be recommended. This consists in grasping the child by the buttocks with the left hand, holding it in such a position that the head will be lower than the body, the thumb and forefinger of the right hand being placed around the neck. The



FIG. 7.—ARTIFICIAL RESPIRATION (INSPIRATION).

left hand then raises the body, bending the latter over, and during this movement the thumb and first finger of the right hand are making pressure against the thorax; this constitutes expiration. The second movement consists of extending the body or straightening it out, the

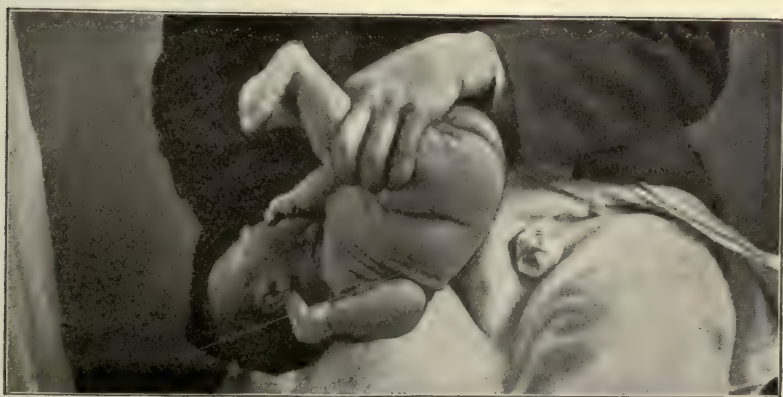


FIG. 8.—ARTIFICIAL RESPIRATION (EXPIRATION).

thumb and first finger of the right hand at the same time releasing their pressure upon the chest-wall; this constitutes the act of inspiration. The arms may be allowed to hang down, or, better, may be partially supported by the other fingers of the right hand and the body of the operator. It is well to have the lap covered with a blanket or a rubber

sheet in order to protect the clothes. The movement should be repeated from eighteen to twenty times a minute. The faradic current is also very useful in many of these cases.

*Mouth-to-mouth Insufflation.*—The air-passages of the child having been cleansed of mucus, the physician places his mouth to that of the child and expires with some force into its air-passages. At the end of each expiration (the child's inspiration) pressure is made on the child's thorax and abdomen, so as to cause the infant to force the air from its lungs. It is unnecessary to close the child's nostrils during its inspiration. Direct insufflation can sometimes be made by passing a soft catheter into the larynx and gently inflating the lungs, either by expiring into them or by the use of the apparatus of Depaue or Ribmont.



FIG. 9.—RIBMONT'S TUBE.

In cases of late asphyxia following non-expansion of the lungs, our efforts should be directed toward exciting more active respiration.

The stupor which sometimes appears is to be treated by application of hot and cold water to the head and body, or some stimulating liniment may be used.

Unfortunately, the dangers of asphyxia do not end with the resuscitation of the child, and this is especially the case in the pallid or asthenic form. In many of these patients the first respiratory efforts are so feeble that only small areas of the lungs are inflated. What expansion does take place occurs near the surface of the lung. Frequently not enough expansion occurs to allow of the aeration of sufficient blood to support life. Atelectasis frequently occurs. In treating the pallid form of asphyxia a good plan is to immerse the infant in a hot mustard bath at a temperature of 110° F. (43.3° C.). A small stream or spray of cold water may be poured on the chest from time to time. In addition to the methods of artificial respiration described, hypodermic injections of minute quantities of stimulants, such as aromatic spirits of ammonia, strychnin, atropin, and brandy may be used with good effect. These patients require careful watching for several days. Artificial respiration and general stimulation should be repeated if necessary.

In the after-treatment the indications are two: warmth and fre-

quent, regular feeding of small quantities of suitable food. Heat at a regulated temperature can be obtained by wrapping the child in raw cotton or wool and placing it in a padded basket near the fire, with one or two bottles of hot water near it and a bath thermometer inserted near the body. A better method is to place the child in the *couveuse* or incubator, the one designed by Auvard, or one of its modifications, being about the most convenient. The apparatus consists of a glass-covered box in which a small basket or pillow is placed upon a false bottom on which the infant lies. Warm air is generated beneath the

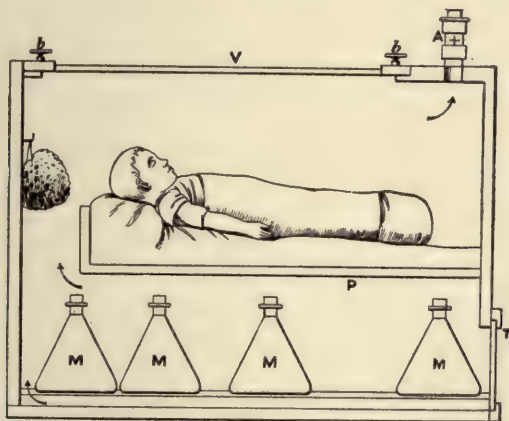


FIG. 10.—DIAGRAM OF TARNIER COUVEUSE.

false bottom by means of cans filled with hot water. The air, admitted at a small door beneath the false bottom at one end of the box, is heated by its passage over the cans filled with hot water, then passes up through the chamber in which the child lies, and escapes out through a tube in the top. In this tube a small revolving fan is sometimes placed to secure better ventilation.

#### THE MANAGEMENT OF INFANTS PREMATURELY BORN

The fundamental principles for the care of prematurely born infants are to be learned from a study of intra-uterine life. The prematurely born infant must be carefully protected from cold and changes of temperature: in other words, it should live in an atmosphere the degree of heat of which is kept as near that of the mother's body as possible. It should receive a plentiful supply of pure warm air, and as the eyes are as yet but incompletely developed, they should be protected from light by keeping the child in the dark. Nourishment must be given by the mouth in the most digestible form possible, in small quantities, and at regular frequent intervals.

*Temperature.*—A prematurely born infant often loses from five to nine degrees of bodily heat in an hour after birth (Eröss). This rapid loss is partly due, in very premature infants, to the almost entire absence of subcutaneous fat and to the greater proportionate surface area of the body (Blacker). Another factor to be considered is that on account of the general feebleness of these infants and their weak digestive powers they cannot produce much heat in themselves.

*Light.*—On account of the imperfect development of the eyes, these should be carefully protected from light until the infant has arrived at full term. This can be done by covering the glass top of the incubator with a piece of dark-green or black cloth.

*Nourishment.*—Every premature infant should be carefully weighed at frequent regular intervals, at first every day, unless it be too feeble. It is only by this means that the increase or decrease in the child's development can be studied. The average daily increase in weight varies with the total weight of the child, and is, of course, less than in full-term children. This increase may amount to from  $1/4$  to  $3/4$  of an ounce a day, and is by no means constant, even if the child be thriving. It must be remembered that these children often remain stationary, as far as weight is concerned, for days at a time; therefore as long as there is no actual loss, no anxiety need be felt. Should a steady decrease in weight occur, the child's life is in danger and a careful investigation must be made for the cause. A very slight loss of weight is sufficient to cause death.

The first indication in the treatment of these infants is to keep them in a warm atmosphere of as nearly even temperature as possible, and this can be best done by the use of the couveuse, or incubator, one form of which has been described.

The temperature of the incubator should at first be kept at from  $85^{\circ}$  to  $90^{\circ}$  F., according to the general condition of the child: thus, if its extremities are cold or its temperature subnormal, the temperature of the incubator must be raised; on the other hand, if perspiration appears on the child's body, this is at once an indication for reducing the temperature of the couveuse. If the child continues to thrive, the temperature of the couveuse must be gradually lowered, so that when the child is removed, it may not be injured by the temperature of the ordinary room, which is about  $70^{\circ}$  F.

It is highly essential that the air which the child receives should be as pure as possible; therefore the incubator should be placed in a well-ventilated room. An upper room considerably above the ground-level will generally answer best. The air entering the incubator should

be slightly moist and well filtered. In hospitals this can be done by having the incubator supplied with outside air passed over water or through moist cotton. In private houses a wet sponge placed in a small rack in the incubator, so that the incoming air can pass through it, will answer the purpose quite well. In the presence of emergencies, a basket with hot water bottles or a wash boiler, similarly equipped, may furnish an excellent temporary incubator. Great care must be exercised to prevent burns, however. It is generally considered best not to bathe a premature infant, at least during the period of its incubator life; instead it should be enveloped, piece-meal, in cotton-wool, which can be changed twice a day or when necessary. Oils and ointments are contraindicated.

The feeding is a most important factor in the treatment of these infants; many of them do well on their mother's milk, and in all cases an attempt should be made to feed the child with this. The milk should be drawn from the breast by a clean breast-pump. If the child is old enough to suck, it may be successfully fed by the instrument devised by Breck. This consists of a graduated glass cylinder holding about nine drams. It is shaped at one end so as to hold a small rubber nipple. The large end is covered by a small "cot" or air reservoir. To fill the tube, the rubber nipple and cot are removed, and the opening at the nipple end closed with a rubber plug. The food is then poured in the large end. In feeding the child the plug is withdrawn and the nipple and cot replaced; the nipple is introduced into the mouth, and by gently pressing the rubber cot the food is slowly forced down the infant's throat without any danger of choking it or the expenditure of its strength. If such an instrument cannot be had, or if the infant be too feeble to suck, it may be fed from a curved medicine dropper or even by a spoon. If the administration of food by the dropper causes vomiting, the child must be fed by gavage. A small soft catheter is introduced into the stomach, and the food poured in by means of a funnel. This method is, however, much more uncomfortable for the child than feeding by the dropper, and should only be used when all other means have failed.

In some cases mother's milk, even when diluted, will not agree

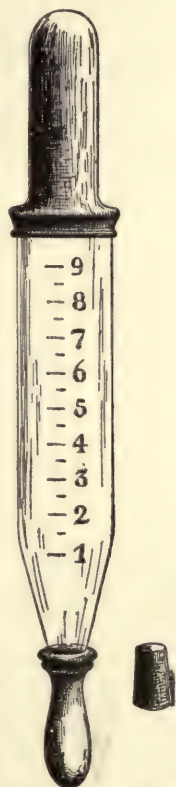


FIG. II.—BRECK'S INFANT FEEDER.

with the infant, this being explained by the fact that up to the end of pregnancy, and, indeed, for some days afterward, the secretion of the mammary glands is colostrum rather than true milk. This colostrum milk is nearly always present at the time of or follows premature birth of the child, and analysis of it shows that it contains an excess of proteids with a relative deficiency of sugar and fat. It is on this that its laxative properties and tendency to cause indigestion are supposed to depend. As a rule, the more premature the birth, the longer the colostrum character of the milk continues.



FIG. 12.—MODIFICATION OF TARNIER COUVEUSE, IN USE IN THE JEFFERSON MATERNITY, PHILADELPHIA.

The stomach of the premature infant is a very small and delicate organ, and its digestive fluids are weak; therefore it is highly important that the food which is put into it shall be such that it can be easily and quickly digested and assimilated. If this is not the case, the child will lose weight and its chances of surviving are much diminished. It is important, therefore, that if the mother's milk, plain or diluted, does not agree from the first, it must be stopped and some easily digested artificial food be substituted for it. Whatever food is used should be given in very small quantities, often not more than a dram at a time, and this must be repeated frequently. The simplest food to use in feeding premature infants is a mixture of equal parts of peptonized milk

and barley-water, and of this a teaspoonful may be given every two hours; but much better, in our experience, is cow's milk, so modified that the amount of proteid shall be exceedingly small. Some of the formulas of Rotch will be found very useful for this purpose. As every infant, no matter how young, is a "law unto itself," every patient must be studied with the utmost care. A given milk formula must be studied in the light of the digestibility of each and all of its elements, and when these are taken up by the child's digestive organs and assimilated, the child thrives; otherwise it will rapidly lose ground. If a formula is not agreeing, as a rule the element, be it fat, sugar, or proteid, which is in excess and which is causing the trouble will show itself by the symptoms it produces, and so the reducing of it is an easy matter. We usually start with a 1.00-4.00-.33 per cent. formula, and to save the baby energy and avoid digestive disturbances, we peptonize this. The baby is fed every one and one-half to every two hours, according to indications. He is given from two to six drams at every feeding, age, weight and hunger determining these quantities. The next formula employed is usually a 1.50-4.00 and .50 per cent.

If the first formula does not agree, the element in excess must be sought for and reduced. If, on the other hand, the food agrees and the infant does not gain in weight as fast as it ought to, a richer food is demanded. As a rule, some modification of cow's milk which will answer all purposes can be made by the mother or nurse at home. A little patience on the part of the doctor and nurse and some knowledge of the physiology of the infant's digestive apparatus will enable them to make up a food which will meet all requirements. The use of condensed milk, boiled milk, or patent foods, as advised by some authors, we must condemn.

If the infant be very feeble, a few drops of brandy may be given every two or three hours as long as necessary, and if cyanosis should at any time appear, oxygen may be administered for five or ten minutes two or three times a day.

## DISEASES IN THE NEW-BORN CHARACTERIZED BY HEMORRHAGE

### HEMORRHAGES IN THE NEW-BORN

**Etiology.**—Hemorrhage in early life may, as in the adult, depends upon an alteration in the condition of the blood itself or upon direct injury to the blood-vessels.

As instances of the first we have the extensive disintegration of the blood, found in syphilitic infants and after the acute fevers, such as typhus, scarlet fever, etc.

Intra-uterine or postnatal infection of the fetus by micrococci may be a cause of hemorrhage.

Hemorrhages from the navel following infection are also instances of this class. Tavel and Quervian have reported a case in which death on the thirteenth day after birth followed an infection of the umbilicus occurring immediately after the child was born. Post-mortem examination in this case showed hemorrhages into the connective tissue, beneath the epidermis, the mucous and serous membranes, and in the kidneys. Direct evidences proved these hemorrhages to be caused by streptococcus infection.

In a second case death occurred from pneumonia which was the result of staphylococcus infection, as was proved by subsequent examination. The hemorrhages had occurred into the parenchyma of all the organs examined.

**Symptoms.**—The symptoms in the majority of cases of hemorrhage are very obscure. Bleeding occurs from the umbilicus and mucous surfaces internally, externally, or both. Subcutaneous hemorrhages (petechiæ) are common. The infant usually ceases to nurse, and is somnolent and pale. Later, convulsions and irregular respirations develop, and the child dies. A proper study of these cases can be completed only by making a postmortem examination of all infants dying of obscure symptoms.

#### APOPLEXY IN THE NEW-BORN

**Synonyms.**—CEREBRAL OR MENINGEAL HEMORRHAGE; ASPHYXIA RUBRA SEU APOPLECTICA; HYPEREMIA CEREBRI TRAUMATICA

**Definition.**—Cerebral hemorrhage occurring in early life. The term is, however, often used to cover all degrees of increased cerebral blood pressure.

**Etiology.**—The most frequent cause is pressure during birth or continued compression of the head by forceps. It is to be recalled, however, that much more harm is done by the undue prolongation of a difficult labor, with its resulting asphyxia neonatorum, than is done by the use of obstetric instruments in skilled hands. It may follow pressure on the umbilical cord during a breech presentation or otherwise prolonged labors.

Apoplexy in the new-born is most generally the result of a venous congestion of the vessels of the pia mater or choroid plexus.

**Pathology.**—Following distention of the cerebral sinuses by asphyxia or other causes, or a rupture in the capillary vessels of the pia mater, the blood is effused into the subarachnoid space. As in the very young the connection between the inner covering of the brain and the cerebral tissue is not intimate, the area of hemorrhage may extend over a large surface or burst into the subdural space, compressing the brain substance—indeed, an actual laceration of the brain tissue may occur, leading to a secondary softening. Engorgement of the superficial cerebral veins may take place: as, according to Gowers, the ascending arteries pass into the ascending veins, and these empty themselves into the superior longitudinal sinus in a forward direction and consequently against the blood current.

Spencer and McNutt have concluded that while in most cases apoplexy in the new-born is seen in children delivered after difficult labors or where the forceps have been used, it sometimes occurs after short and easy labors, and one of the writers has recorded a case in which an infant born in breech presentation and by an easy labor presented the following symptoms: shortly after birth the breathing became irregular, and, later, difficulty in swallowing, convulsions, left-sided hemiplegia, and rapid emaciation made their appearance.

The length of the child's life was twenty-two days. At the autopsy it was found that a clot covered the right hemisphere, this clot being gelatinous and firm and of a dark color. Not only the convolutions beneath it were in part destroyed, especially in the frontal and parietal regions, but also the brain tissue covering the ventricle, while the sites of the corpus striatum and optic thalamus were occupied by a reddish-brown clot mixed with softened brain substance.

The blood may be extravasated in points here and there over the entire brain, or may only occupy a part of the organ. In other cases extravasation may take place in one or perhaps two of the cavities in the same manner as in ordinary apoplexy. In the first form there is little laceration or injury to the brain substance. The cerebral tissue surrounding the hemorrhagic points sometimes preserves the normal appearance, being white and firm. Occasionally, however, it may present a reddish or yellowish appearance, being softened to a depth of a line or two. When the hemorrhage occurs into a cavity, in the same manner as in apoplexy of adults, the nerve-fibers are generally torn and separated. In these cases there is sure to be more or less compression of

the surrounding brain substance. Unless the disease be of long standing, the cavity contains a dark, soft clot, bathed with serum. The brain substance in the immediate vicinity is softened.

Partial or complete asphyxia will cause intense congestion and engorgement of the cerebral vessels, being accompanied or followed by hemorrhage on or into the brain tissue in many instances, and, as a result, there will frequently be found a chronic meningo-encephalitis, a sclerosis, or an atrophy of a part of the brain, which in a certain percentage of cases may be followed by chronic paralysis or deficient mental development.

**Symptoms.**—The infant, which at the moment of birth may evince no particular symptoms, in a few hours becomes cyanosed. More or less projection of the eyeballs may be present and the tongue possibly be protruded. If the hemorrhage is at the base of the brain or is severe, the cardiac action and respirations become weak and irregular, and the child dies in collapse. If the effusion is small or is in the cortex, or if simple hyperemia without rupture is present, the child may recover. It should not, however, be forgotten that permanent paralysis, convulsions, or even idiocy may follow such a condition.

Davis cites a case in which an infant delivered with axis-traction forceps without difficulty displayed symptoms of progressive feebleness of respiration. Failure to nurse and apparent exhaustion caused death in thirty-six hours after birth. A postmortem examination showed the tissues of the scalp to be intensely congested, but no gross lesion, either rupture or fracture, was present. The cortex of the cerebrum was filled with punctate hemorrhages, and over the point of greatest convexity the brain substance was materially softened. Virchow and others have shown that the blood-vessels of the infant's brain are thin and small, and most readily injured by abnormal pressure.

**Treatment.**—The indications for treatment are mostly prophylactic, and care should be taken to prevent excessive birth pressure by prompt deliverance of the child by forceps or suitable operative procedure. There is much more danger in the continued pressure on the child's head by the maternal parts during long labor than by a skilful forceps delivery. On the other hand, undue pressure on the fetal head by the forceps or the forcible rotation of the same should be avoided. The work of Cushing and others has opened a hopeful field of work for the brain surgeons. When convulsions (unilateral or bilateral), pareses and paralyzes proclaim the existence of intracranial hemorrhage, the patient should be given the benefit of the doubt and trephined. Even if there be great risk attached to opera-

tion at this age, a possible future of paralysis, epileptiform seizures and mental deficiency, makes the risk seem more than justified.

### HEMORRHAGES FROM MUCOUS SURFACES

**Etiology.**—Hemorrhages from various mucous membranes of new-born infants result most frequently from malnutrition and its consequent anemia or from the hemorrhagic disease of the new-born. They may also be caused by congestion of the pelvic organs, due to the sudden cessation of the flow of blood through the umbilical arteries. Syphilis is a frequent cause, more frequent than most physicians suppose. Infections are also commonly responsible for such hemorrhages.

The most common site of such hemorrhages is from the mucous membrane of the vagina in female infants, although it may occasionally occur from the rectum or mouth, and, rarely, from the nose.

**Treatment.**—When the hemorrhage is the result of simple congestion in a robust, hearty child, no treatment is required. When the infant is anemic, it is well to employ minute doses of arsenic, careful regulation of the nursing, and inunctions of olive or cod-liver oil, combined with soap liniment in the proportion of about three parts of the former to one part of the latter. Mercury by inunction should always be used when there is the slightest suspicion of lues.

When the bleeding surface can be reached, applications of a solution of boric acid or a hot mixture of creolin and water, one dram to the quart, are useful. In several instances we have seen calcium lactate appear to exert a wonderful influence when administered in doses of from  $1/2$  and 1 grain. Injections of gelatin may also be tried. Adrenalin locally applied, sometimes acts most promptly.

In view of the recent work of Lambert, Welch, Schloss and Commiskey and others, all methods of treating the hemorrhagic diseases of the new-born, must pale into insignificance when compared to the injection or transfusion of human blood, or the injection of human serum. Welch saved twelve successive cases, and the only patient by Schloss and Commiskey was moribund when injected. We agree with the last-named authorities that the withdrawal of blood from a vein of the donor, and its immediate injection into the infant furnishes at once the simplest and the safest method of procedure. The procurance of serum takes time, and transfusion may prove dangerous because of the tediousness of the operation. From 10 to 20 c.c. of blood may be withdrawn into a sterile syringe and immediately injected into the subcutaneous tissue of the infant. In the further discussion of hemor-

rhagic diseases in the new-born, we shall not mention these methods, but it is to be understood that they should be given a trial in all serious cases.

### CAPUT SUCCEDANEUM

**Synonyms.**—SUPPLEMENTARY HEAD; SPURIOUS CEPHALHEMATOMA; SUBAPONEUROTIC CEPHALHEMATOMA; KOPFGESCHWULST

**Definition.**—A tumor upon the presenting part of the fetus, the result of serosanguineous infiltration under the skin and subcutaneous tissue, due to pressure.

The tumor occurs upon that portion of the presenting part of the child which is itself not subjected to pressure.

The size of a caput succedaneum increases generally in proportion to the length of the labor. The color of the tumor is bluish red, and there is no fluctuation or pitting on pressure.

**Etiology.**—Continued pressure upon that portion of the fetal skull which receives the greatest impact of force during descent and rotation, temporarily checks the free circulation of the blood and lymph through the tissues of the scalp and fascia. On the opposite side of the vertex there remains a portion of the scalp which endures less pressure from the bony pelvis, and it is here that the blood and lymph of the scalp are prevented from circulating through the other side of the fetal head by pressure, and thus accumulate, distending the tissues of the side least pressed upon. Consequently, we find the tumor generally on the side opposite to that which is actually engaged in the pelvis in the first stage of labor.

The *situation* of the caput succedaneum will sometimes give a clue to the position occupied by the child in the uterus: thus, in the case of an infant occupying a position with the vertex to the right anterior half of the mother's pelvis and the fetal back to the mother's right, the caput succedaneum will be found on the left parietal portion of the child's head.

In shoulder presentation the tumor is found on the presenting shoulder.

**Varieties and Pathology.**—In cases in which the labor is very protracted and the head subjected to long-continued pressure, the caput succedaneum may cover *both* parietal bones, and this fact is regarded as one of the diagnostic features between it and true cephalhematoma.

Occasionally two tumors, a primary and a secondary, may be

found, and in such cases the first tumor is formed in the manner previously described and the second is due to pressure after the head is rotated on the floor of the pelvis, the presenting part being detained for a considerable time in this position.

**Treatment.**—In the majority of cases caput succedaneum needs no treatment. When the tumor is very large, some stimulating evaporating lotion, such as solutions of chlorid of ammonia, camphor and alcohol, or cold cream, may be applied on cotton, which should be held in place by a firm bandage. As a rule, the tumor need occasion no further solicitude.

If the tumor persists, some authorities advise either aspiration or incision, after which pressure, by means of a pad of salicylated cotton and a bandage, should be applied. If, as sometimes happens, the tumor becomes infected and an abscess forms, it should be opened under strict antiseptic precautions and treated in the usual way.

## CEPHALHEMATOMA

**Synonyms.**—THROMBUS NEONATORUM; ECCHYMOMA  
CEPHALHÆMATOME

**Definition.**—An elastic, non-fluctuating tumor of hemispheric form, occurring usually on the scalp and increasing in size after birth.

**Etiology.**—The causes of cephalhematoma are obscure, and the literature of the subject contains many conflicting statements regarding its etiology.

According to some authorities, cephalhematoma is in part caused by asphyxia, during which there are increased tension in the cranial veins and an altered condition of the blood, allowing extravasations.

Pressure would seem in some cases to be a cause, as might be inferred from the situation of the tumor—namely, on the right parietal bone; but the literature of the subject contains the reports of a number of cases in which the tumor occurred upon parts which had not been subjected to pressure during birth, and one case is recorded in which the tumor was seen on the head of a child delivered after Cesarean section.

Virchow believes that the pericranium is formed by a proliferation of the inner layers of the periosteum. When separation of the latter from the bone by an extravasation of blood occurs, the bone-producing layers of the periosteum are still formed, but are prevented by the blood-clot from uniting with that portion of the bone for which they

are intended; they therefore join themselves to the bone at the border of the extravasated clot where the bone is yet attached.

The cause of cephalhematoma has also been ascribed to traumatism and to alteration in the blood itself, this alteration being due to malnutrition.

Regarding the frequency of cephalhematoma, it is found more often in males than in females. It occurs about once in every 200 births.

Hennig noticed that it was situated fifty-seven times over the right parietal bone, thirty-seven times over the left, and in twenty-one cases it was found over both; in seven cases it was over the occipital bone, three times it was noticed over the frontal, and twice over the temporal.

Cephalhematoma never extends beyond the borders of the bone upon which it is situated.

**Symptoms.**—The tumor usually appears on the third or fourth day after birth. In appearance it presents no discoloration of the



FIG. 13.—SECTION OF CEPHALHEMATOMA.

surface and is without fluctuation. In size it varies from that of a walnut to that of a hen's egg. Usually there is no discoloration of the scalp around its borders. Little or no sensitiveness exists in the tumor.

The swelling usually remains about a week after its appearance. For the first few days it continues to increase in size and then slowly diminishes.

A few days after its development a distinct and well-defined ridge will be found at its circumference, this ridge being due to deposits of bone made by the periosteum.

Examination of this ridge may give the impression that a defect exists in the bone upon which the tumor occurs, and that the tumor is protruding through a perforation in the cranium.

After the decrease and disappearance of the tumor, the bony ridge may remain for a time, but finally disappears, leaving no trace behind.

**Pathology.**—The usual situation of the tumor is on the right

parietal bone; a number of cases, however, have been reported in which the cephalhematoma was found in other situations.

Hofmohl observed it to be bilateral in twenty-six cases, and in each of these the fontanel lay as a deep sulcus between the tumors. A classification of cephalhematoma based on its situation has been made by various authors. Ashby and Wright differentiate it as follows:

EXTERNAL CEPHALHEMATOMA. . .	{ 1. Subaponeurotic, spurious cephalhematoma, or caput succedaneum.
INTERNAL CEPHALHEMATOMA. . . . .	2. Subperiosteal, the true cephalhematoma.
MENINGEAL HEMORRHAGE. . . . .	3. Subcranial.
	4. Subarachnoid.

It would seem to the authors, however, not only from their own experience, but also from a review of the literature on the subject, that cephalhematoma is usually described as that form of blood tumor beneath the periosteum and external to the cranial bone—the subperiosteal of the above classification, and it is the pathology of this form which is here described. Other forms of hemorrhage within the cranium belong more properly under the classification of “intracranial hemorrhage.”

Macroscopically, sections of cephalhematoma show that an extravasation of blood has taken place between the periosteum and bone, the surface of the latter being roughened.

Some thickening, the result of inflammatory irritation, occurs around the margin of the tumor where the pericranium is attached, and it is at this point that the shell-like deposit of bone occurs.

Kirk describes the formation of these tumors as follows:

The principal bones forming the cranial vault are developed in membrane or fibrous tissue. This membrane later consists of two layers, an external fibrous and an internal cellular—osteogenetic or the true bone-forming layer. If a thick metal plate be inserted beneath the periosteum during its formation, it will soon be covered by an osseous deposit; but if placed between the fibrous and the osteogenetic layer, the plate will not be covered with bone-cells, thus showing that the osteogenetic layer is only capable of developing bone from its lower surface. When extravasation of blood takes place, as in the true form of cephalhematoma, the osteogenetic layer is pushed away from the bone, but continues its power to develop bone-cells, and this deposit is increased around the area of limitation of the tumor—that is, the point at which the periosteum covering the tumor joins that which covers the remainder of the cranium—thus forming the hard bony shell which surrounds the tumor.

**Diagnosis.**—Cephalhematoma may be differentiated from caput succedaneum, hernia of the brain, or its membranes, craniotabes, and cranial angiomata. From caput succedaneum it can be distinguished by the fact that cephalhematoma bears no relation to the difficulty of the labor; on the contrary, it frequently does not appear until some days after birth. In cephalhematoma we find a fluctuating center to the tumor, but it lacks the boggy feel of caput succedaneum. There is no discoloration of the scalp in the former, as would be found in the latter. Cephalhematoma also never crosses a suture, and is surrounded by the ring of bone before mentioned.

From hernia cerebri it can be distinguished by the absence of fluctuation in protrusions of the brain. In hernia there will be pulsations which are synchronous with the child's heart-beat.

*Hernia* also enlarges when the child cries, and always shows itself between two bones or in the region of a fontanel.

*Craniotabes* can be diagnosticated by the softened patches which occur in the skulls of children affected with rickets, or syphilis. These lack the swelling and clearness of outline of a cephalhematoma and no fluctuation is present.

In *vascular tumors* of the scalp there is discoloration of the parts and absence of fluctuation in the center; there is no bony growth around the tumor, and the swelling bears no relation to suture or fontanel.

**Prognosis.**—Unless some systemic weakness is present, the prognosis is good.

**Treatment.**—In the largest number of cases cephalhematoma will disappear without any treatment whatsoever. In some instances where the tumor is not large, shaving the hair and painting the growth with collodion or some evaporating lotion is recommended. In regard to aspirating or opening the tumor, the opinions of authors differ: the former treatment may be followed by sepsis, and the danger of severe hemorrhage may present itself after incision of the tumor when the growth connects with an extravasation of blood inside the cranium.

#### HEMATOMA OF THE STERNOCLEIDOMASTOID

The most common cause of this tumor is injury to the neck of the infant following an attempt at forcible extraction of the head in a breech presentation.

The danger of the injury is increased when the infant is partially asphyxiated, in which case the muscles lose their tone, the vessels weaken, and on this account the escape of blood is more apt to occur.

The direct cause of such a blood tumor is the laceration of a vessel and consequent hemorrhage into the sheath of the sternocleidomastoid muscle of one side.

Microscopic examination will show effusions of blood with rupture, more or less extensive, of the muscular fibers.

Jacobi considers prolonged extraction with forceps a frequent cause of this abnormality.

**Symptoms.**—The swelling may appear on the neck in from a few days to a week after birth, and on examination a tumor varying in



FIG. 14.—HEMATOMA OF THE STERNOCLEIDOMASTOID.—(From a patient in the Philadelphia Polyclinic.)

size from that of a walnut to that of a hen's egg is found in the upper part of one sternocleidomastoid muscle, the right side being the most frequent site of the hemorrhage. The outline of the mass is somewhat irregular, and if existing for some time, the growth may be cartilaginous. The duration of the tumor is from one to two months, after which time it slowly disappears. In one case, studied by one of us, the acromial end of a dislocated clavicle closely simulated this affection.

In some cases chronic torticollis may result from such injuries. Paralysis of the arm on the injured side is occasionally seen. No especial treatment is needed in the majority of cases.

## UMBILICAL HEMORRHAGE

**Etiology.**—Hemorrhage from the umbilicus may result from slipping of a ligature attached to the stump of the cord or by the ligature cutting through the umbilical arteries or veins. Compression of the vessels by the ligature may not be sufficient to stop oozing of blood because of excess of Wharton's jelly, or the stump of the cord may be too short to hold the ligature. Bleeding from the navel is also a symptom found frequently in children suffering from asphyxia or atelectasis, from hemophilia, acute fatty degeneration, acute hemoglobinuria, or from syphilis. Pyogenic infection of the umbilical stump is also a frequent cause of hemorrhage. Again, umbilical hemorrhage occurs in cases of marked jaundice, particularly when that symptom is dependent upon congenital stenoses or *atresias* of the biliary passages.

**Treatment.**—The prophylaxis consists of the careful tying of the cord with an aseptic ligature after it has been washed with some suitable antiseptic solution. The treatment of the stump consists of inclosing it in a mass of gauze or absorbent cotton, dusted over with one part of salicylic acid or boric acid, to three or four parts of starch powder. Some authorities completely amputate the umbilical cord at the present day; while others wipe it off with alcohol and enclose it in a sterile gauze dressing which remains *in situ* until the stump falls off. Acetanilid is dangerous, one of us having seen acetanilid poisoning from the use of one part of acetanilid to five parts of an inert powder.

The stump should be placed on its upper side, with its dressing held in place by a moderately firm abdominal band. When umbilical hemorrhage results from pathologic causes, such as have been previously mentioned, attempts should be made to tie the bleeding vessels by means of a silk ligature or two sterilized surgical pins, the latter being passed through the stump at right angles to each other and a ligature wrapped around the pins in the form of a figure eight. Various styptic applications may be indicated, and pressure by a cotton pad and tight binder should be made.

The **prognosis** of umbilical hemorrhage resulting from any form of constitutional disease is usually far from good.

## GASTRO-INTESTINAL HEMORRHAGE

**Etiology.**—Hemorrhage from the stomach or intestines may arise from the passage of blood through the intestines, blood having been swallowed in nursing from a fissured nipple. Such blood is either

vomited or passed through the bowels as black or brownish-black masses.

Hemorrhage into the bowel, the result of long-continued pressure during birth, may cause evacuation of similar masses in the stools.

Perforation of a blood-vessel in the duodenum through an intestinal ulcer is a somewhat rare cause of this form of hemorrhage.

Those hemorrhagic diseases of the new-born due to antepartum infections may also be a cause of gastro-intestinal hemorrhage, and when originating from this source, the bleeding appears in the first day or two of life. Purpura and syphilis have also been given as causes.

Occasionally a slight bleeding from the intestine may arise without any apparent cause. Possibly this hemorrhage may be produced by a congested condition of the abdominal organs due to the change in the circulation at birth. An initial chilling sometimes precedes the appearance of melena.

The **symptoms** are those of internal hemorrhage at any time of life. The child becomes restless, pale, the extremities are cold and the fontanels are sunken. The child will vomit, and with the vomited matter brownish-black masses of blood will be found.

The same characteristic masses will be found in the discharge from the bowels. The abdomen becomes dull and tumid on percussion.

**Pathology.**—In many cases a postmortem examination will reveal nothing but a simple congestion of the gastro-intestinal mucous membranes. In other instances ulceration of the stomach or intestines will be found.

**Treatment.**—The treatment should be directed toward checking the hemorrhage, when this is possible. For this purpose ergot or ergotin, the latter in doses of from  $\frac{1}{4}$  to  $\frac{1}{2}$  of a grain, may control the bleeding. The drug may be given mixed with simple syrup of mucilage. Tannin or gallic acid in small doses in syrup of rhatany is advised by some. Calcium salts and gelatin should be used in desperate cases.

Occasionally good results follow gentle irrigation of the bowels with a solution of sodium chlorid  $\mathfrak{Z}$ i to the pint at a temperature of  $110^{\circ}$  F. ( $43.3^{\circ}$  C.), the injection being given from a fountain syringe.

## MELENA IN THE NEW-BORN

**Definition.**—A malignant form of hemorrhage from the stomach or intestines, occurring in the new-born.

**Etiology.**—The hemorrhage may be due to a gastric or enteric

ulcer; to thrombosis resulting from embolism in the vessels of these organs. Congenital weakness of the vessels of the stomach or duodenum and persistence of the ductus arteriosus have been ascribed as causes. Most frequently, however, it is dependent upon a septic cause, or upon syphilis. As previously mentioned, it has occurred in babies who have been subjected to an initial chilling.

**Symptoms.**—The symptoms are vomiting of blood or its passage in the stools. This condition is accompanied by rapid loss of flesh, failure to nurse, and continued hemorrhage, until the child dies of collapse. The attack usually lasts from one day to a week. While the prognosis is grave, cases occasionally recover.

**Treatment.**—The treatment consists of the use of warm antiseptic rectal irrigations, and the administration, by the mouth, of ergot or some astringent remedy. Mercury is to be given in all suspicious cases, and the calcium salts are to be used internally.

## DISEASES CHARACTERIZED BY JAUNDICE

### ICTERUS IN THE NEW-BORN

**Synonyms.**—YELLOW GROOM; INFANTILE JAUNDICE; GELBSUCHT; ICTÈRE

Usually about the third to the fifth day of life a certain amount of yellowish discoloration of the skin appears. This first manifests itself in the face, and quickly spreads to other parts of the body. It continues for five or six days and gradually disappears. The urine during this time assumes a saffron color, and examination will reveal an excess of bile pigment. This condition is known as physiologic jaundice. It occurs in from 60 to 80 per cent. of infants, and is rather more common in children born in hospitals than in those seen in private practice.

**Etiology.**—The following theories have been ascribed as causes of jaundice in the new-born infant:

Following birth a rapid destruction of blood-corpuscles takes place, this producing an excess of bile pigment. The jaundice then is said to be of hematogenous origin. It is also supposed that a certain amount of blood from the portal vein, owing to a patulous condition of the ductus venosus, passes into the general circulation without being acted on by the liver. The swelling of Glisson's capsule is also given as a cause; this swelling commences at the umbilical vein and prevents the discharge of bile through the hepatic vessels. Other causes, such as alteration of the blood pressure at birth and congested condition of the skin, have been given.

**Symptoms of the Simple Form.**—A slight yellowish discoloration of the face appears about the third or fourth day, and in the course of a few hours the entire body assumes a yellowish tint.

Not infrequently the pigmentation may extend to a slight extent to the conjunctiva or sclerotic.

**Treatment.**—No especial treatment is required other than some mild laxative, such as a half teaspoonful of olive or castor oil or the following formula:

R̄. Hydrarg. chlor. mite.....	gr. 1/40
Pulv. ipecac.....	gr. 1/10
Sodii bicarb.....	gr. ij.

SIG.—To be given every two or three hours.

### MALIGNANT JAUNDICE IN THE NEW-BORN

Under the title of morbid or malignant jaundice in the new-born infant may be described the discoloration of the skin and coexisting symptoms resulting from retention in the blood of various bile products (hepatogenous jaundice). This retention is due to stricture, congenital or acquired (stenoses or complete atresias), to catarrh of the gall-ducts or gall-bladder, duodenal catarrh, defective hepatic circulation, asphyxia, melena, Winckel's disease, long-continued birth pressure, syphilis, or a continued exposure to damp, cold, or impure atmosphere. Jaundice is also a frequent symptom of septic infection in new-born children.

**Symptoms.**—In conjunction with the symptoms of any of the diseases given as causes, we find the ordinary jaundice in the new-born continuing beyond its usual period of duration. These symptoms are accompanied by increasing drowsiness, subnormal temperature, and failure to nurse. The stools are black and tar-like. Unless amelioration of the symptoms occurs, death follows on about the tenth day.

**Diagnosis.**—This is easily made from the color of the skin, conjunctivæ, character of the stools, and, in the malignant form, the general symptoms. Of great importance is the differentiation of the disease causing it. When the jaundice arises from acute fatty degeneration or from hemoglobinuria (Winckel's disease), the blood changes (see description of these diseases), purpuric patches in the skin, hemorrhages, or cyanosis will aid in the diagnosis.

When obstruction to the duct of the gall-bladder (hepatogenous jaundice) produces the disorder, the discoloration is intense and no bile can be found in the stools.

**Treatment.**—The treatment must be directed to the cause. Action of the ducts and intestine should be stimulated by small doses of calomel combined with phosphate of soda, chalk, or lime. Attention must be paid to the skin, to increase as far as possible its activity.

The infant should be kept in a warm, pure atmosphere and should be fed as much as possible on breast milk or the best artificial substitute that can be obtained.

The action of the kidneys must be increased as much as possible by small quantities of hot boiled water given regularly.

### ACUTE HEMOGLOBINURIA OF THE NEW-BORN

#### **Synonym.**—WINCKEL'S DISEASE

This disease was first described by Winckel, who reported the results of an epidemic in which twenty-three cases were affected by the disease in the Foundling Hospital in Dresden, in 1879. Chanin, in 1873, and Bigelow, in 1875, also described this affection.

**Pathology and Symptoms.**—The disease is characterized by a swelling of Peyer's patches and the mesenteric glands. In the cases reported by Winckel the pyramids of the kidneys were colored a blackish-red with stripes of hemoglobin coloring. The liver and other viscera were affected by fatty degeneration. "Hematogenic icterus is present, the hemoglobin being extensively changed into bilirubin. The urine is reddish-brown in color and contains epithelial casts, hemoglobin, and micrococci." It is passed in small quantities and after much straining. The first symptom noticed is a bluish tint of the face, body, and limbs—cyanosis. A little later there usually appears some yellowish discoloration of the skin of the entire body. These symptoms begin about the fourth day and progress rapidly. Diarrhea and vomiting soon make their appearance, and in a short time the child refuses to nurse. The duration of the disease is usually about two days, the child dying in convulsions or collapse. The mortality in Winckel's cases was nineteen out of twenty-three.

### ACUTE FATTY DEGENERATION IN THE NEW-BORN

#### **Synonym.**—BUHL'S DISEASE

The **etiology** is obscure, although in some instances the origin has been ascribed to a condition of lowered vitality in the mother during pregnancy. Apparently the disease begins in the latter portion of

gestation. Asphyxia has been given by some as a probable cause and by others as a result of the disease.

**Pathology.**—The pathologic changes seem to consist of a general parenchymatous inflammation of all the organs. Small hemorrhagic patches are found in the various viscera, and some of these organs are found infiltrated with blood and bile. Microscopic examination of the tissues of the various internal organs, and particularly of the liver, kidneys, and heart, will show a state of acute fatty degeneration.

**Symptoms.**—The disease usually appears in from the first to the sixth day of life, the child becoming jaundiced or pale. Hemorrhages occur from the intestines or umbilicus. Petechial patches will be found under the skin and mucous membranes, particularly that of the mouth. Actual hemorrhages may also take place from the various mucous surfaces. There is more or less cyanosis, and actual asphyxia may be produced by fat emboli being washed into the pulmonary circulation. Dropsy, general or local, will be seen in many cases.

The **treatment** consists of stimulating the patient as much as possible and checking the hemorrhage; for the latter purpose ergot or tannic acid in suitable doses is to be given. In the majority of cases the child dies notwithstanding all our efforts in its behalf.

## DISEASES PRODUCED BY SEPTIC INFECTION

### GENERAL SEPTIC INFECTION OF THE NEW-BORN

**Etiology.**—Septic infection in the new-born may arise either from antepartum or postpartum causes. As antepartum causes may be mentioned gonorrhœal and operative infection of the mother's genital tract by streptococci, staphylococci or the colon bacillus. The most frequent causative factor is the entrance of infective micro-organisms through the granulating surface left by the stump of the umbilical cord after the latter has fallen off. From this origin an inflammation of the arteries and veins results, and subsequently thrombi and infiltration of the surrounding tissues follow. The infection usually travels along the course of the umbilical arteries within the abdomen, and later frequently involves the bladder and tissues immediately surrounding it. During the progress of the infection within the body the umbilical scar may remain open, or, as is not uncommonly found, may close and heal, there being nothing left but a small ring of inflammation surrounding it. Weber and Runge have pointed out that in those cases in which infection has occurred through the umbilicus the tis-

sue around the artery is first involved after the infection has traveled within the abdomen. The iliac vessels and retroperitoneal connective tissue are usually not attacked.

As a result of general septic infection peritonitis and metastatic abscesses may appear in the abdominal viscera, or the joints may become involved and arthritis follow. The infection may also enter by the mouth producing in the beginning various forms of septic stomatitis, and occasionally by the tonsils, nasal mucous membrane (septic coryza) and by intrusion into the air passages causing septic pneumonia. From swallowing infected mucus the gastrointestinal tract may be attacked, producing septic diarrhœa (intestinal sepsis). Direct infection through the umbilical ring is, however, by far the most common. In two-fifths of the reported cases Runge observed pneumonia or pleurisy followed by small metastatic abscesses.

When the case has a fatal termination, death usually results from pneumonia or pleurisy, although pericarditis is not an uncommon cause.

**Symptoms.**—In antepartum sepsis, in those cases in which the child is born alive, death usually occurs in a few days from interstitial pneumonia or fatty degeneration. When the child dies before birth, the skin will be found macerated and effusions of bloody serum occur in various cavities of the body. Patches of ecchymosis will be seen in the peritoneum, pericardium, and pleura.

The symptoms of postpartum infection usually begin with a ring of inflammation around the umbilicus; this is often followed by ulceration (omphalitis). In quite a number of cases this inflammation may subside and the umbilical scar will appear to be partially or completely healed. The infant will, however, have a fever ranging from 101° F. to 103° F. Anorexia appears and the child refuses to nurse. The usual jaundice following birth, instead of disappearing on the third or fourth day, will continue to increase, the stools remaining dark and tar-like. Some distention of the abdomen, with general symptoms of peritonitis, soon manifest themselves, the child holding its legs and thighs constantly flexed. The breathing is thoracic in character and is rapid. In some cases ulceration of the mouth, pharynx, intestines, bones, or joints may be observed. Emaciation is rapid and progressive; vomiting and diarrhea sometimes appear. The inflammation of the larynx may result occasionally in actual croup (septic croup). Numerous cutaneous eruptions may also develop in the course of the disease.

When death occurs, the immediate cause is usually convulsions or exhaustion, pleurisy, or pneumonia.

The **prognosis** in septic infection of the new-born is distinctly bad.

**Treatment.**—The best preventive treatment of septic infection in the new-born lies in the careful attention to the umbilicus from the moment of section of the umbilical cord until the time that it has fallen off and the wound is completely healed. It should be remembered that the site of the umbilical cord is always an absorbing surface, through which septic micro-organisms may gain entrance to the child's body, and therefore as much care should be directed to the antiseptic dressing of the cord-stump as would be exercised in dressing any other wound. During the process of mummification of the cord-stump it should be kept covered with some drying antiseptic powder. It is of great importance that the cord-stump should be kept dry. When a drop or two of pus appears in the umbilicus after separation of the cord-stump, the folds of the umbilical scar should be carefully mopped out with a saturated solution of boric acid or hydrogen peroxid, applied on a small piece of cotton.

The superiority of the latter antiseptic lies in the readiness with which it passes between all the folds and crevices of the umbilicus and removes all foci of infection.

Intra-uterine infection should be guarded against as far as possible by careful attention to the mother's health during gestation. Various constitutional diseases, such as syphilis or gonorrhea, should be suitably treated, and all sources of infection from irritating vaginal discharges should be removed by the use of antiseptic douches and cleansing the external genitals during the last week of pregnancy. The constitutional treatment of an infant suffering from septic fever is the same as the treatment of sepsis in the adult, remembering, of course, the age of the patient. The high temperature should be reduced by cool sponging and possibly some alcohol, the latter in doses suitable for the age and condition of the child. Minute doses of strychnin and quinin are also of use.

## OMPHALITIS

**Definition.**—An inflammation of the navel itself or of the surrounding parts.

**Etiology.**—In the majority of cases omphalitis is of septic origin, although occasionally its cause may be doubtful. Syphilis is a frequent cause. It may confine itself to the umbilicus and immediately surrounding tissues, or may spread and involve nearly the whole of the abdominal wall, either superficially or throughout its entire thickness.

The disease begins in the second or third week after birth and may continue for some time. Unless the inflammation extends to the peritoneum, the prognosis is fairly good. In some perfectly normal infants separation of the cord is followed by a slight irritation; this is particularly the case where undue friction or any form of local irritant has been applied. This condition is known as *excoriation* of the navel.

Occasionally after detachment of the cord a serous discharge is noticed; this may exist for some time and is known as *blennorrhagia*.<sup>\*</sup> The site of the umbilicus may also be affected by a *croupous* or *diphtheric* exudate.

The **treatment** of all these forms of inflammation of the umbilicus consists of absolute cleanliness and attention to the rules for prevention before mentioned. When abscesses form, they should be opened and treated antiseptically.

#### TETANUS IN THE NEW-BORN

**Etiology.**—Tetanus in early life, like the disease in adults, is produced by infection with the tetanus bacillus. The usual site of entrance is the umbilicus before the scar is completely healed. Soiled dressings or general uncleanness are the means of transmission. The disease usually appears about the ninth day of life; hence the name "nine-day fits" has occasionally been applied to the disease. It may not, however, appear until the fifteenth day, which has been described by West as the limit at which we usually see the disease. Cases have been reported as early as the third day.

Tetanus in infancy may occur in any part of the world, the largest number of cases being found in warm climates. It may arise sporadically or in epidemics; thus we have reports of the serious epidemics occurring in the island of St. Kilda,<sup>1</sup> and of that occurring in the island of Heimacy, off the coast of Iceland, which took place early in the present century.<sup>2</sup> It seems probable that, as the knowledge of the use of antiseptics, especially as applied to the dressing of the cord-stump in new-born infants, is understood and practised throughout the world, these epidemics of tetanus neonatorum will decrease or almost disappear. The value of the antiseptic dressing of the umbilicus was clearly demonstrated in the epidemic occurring on the island of St. Kilda, where the mortality of cases occurring previous to the introduction of asepsis was 100 per cent.; the number of cases affected decreased after its introduction to *nil*.

<sup>1</sup> Turner, "Glasgow Med. Jour.," 1895, No. 3, p. 161.

<sup>2</sup> Snowman, "Brit. Med. Jour.," 1895, vol. II, p. 132.

**Symptoms.**—The first phenomenon noticed is inability to nurse because of spasms of the muscles of the jaw and face generally; this is known as *trismus*. The facial spasm is soon followed by a similar condition arising all over the body, the attacks increasing rapidly in severity and length of continuance. The face has the peculiar expression described under the name of “*risus sardonius*.” There is also frequently associated with the disease a peculiar whining cry. The climax of the malady is generally reached in twelve hours, and when the child dies, it is in spasms or coma. The convulsions, like those of tetanus in the adult, are increased by cold or by noises. The entire course of the disease is usually about two days.

The prophylactic treatment consists of observing the rules before mentioned in dressing the cord. For the treatment of the convulsions hydrate of chloral, chloroform, and alcoholic stimulants give the best results. Antitetanic serum should be used, though in very acute cases, it may do little good. Should one of us see such a case at the present time, he would inject the serum directly into the spinal canal. Opium, cannabis indica, belladonna, and bromid of potassium have been recommended. Warm baths and ice applied to the spine have occasionally been of use. The prognosis is exceedingly grave.

#### INSPIRATION PNEUMONIA

**Etiology.**—This disease is usually caused by inspiratory efforts on the part of the child, due to pressure on the umbilical cord during a prolonged labor. It is most commonly found in those cases in which the vaginal secretions of the mother have been rendered septic by a preexisting gonorrhea or endometritis.

The type of pneumonia is usually lobular, and is very dangerous to life. The treatment should be prophylactic, care being taken that the vaginal secretion in the mother is rendered aseptic by douches. It must be remembered in the treatment of this disease that the condition is of septic origin, and therefore the principal indications are to sustain the patient by the use of tonics, alcohol, etc. The general treatment of the child is the same, as that of any other form of pneumonia.

#### SCLEREMA

**Definition.**—By the term sclerema we understand a hardening of the skin and subcutaneous cellular tissue and fat. The condition is accompanied by a lowering of bodily temperature.

**Etiology.**—Sclerema may be congenital or acquired; in the latter case it is most frequent in feeble children—those prematurely born or who are syphilitic. It frequently follows exhausting diseases, such as various forms of diarrhea. The disease is most commonly found in hospitals and foundling asylums, particularly where many children are crowded together. It is more common in Europe than in America. The principal cause would appear to be extreme feebleness with continued lowering of the temperature, and, in consequence, a hardening of the subcutaneous fat. Atelectasis is frequently an accompanying condition.

**Symptoms.**—The first symptom noticed is the hardening of the skin, which usually begins in the lower extremities and spreads upward, affecting, in the order given, the trunk, the upper extremities, and the face. It is especially marked in the thighs, buttocks, back, and cheeks. The hardening may be universal or affect only circumscribed areas. The skin may be smooth or lobulated. The skin changes to a dirty yellow or bluish-yellow color, is hard, does not pit on pressure, and seems to be closely attached to the subcutaneous tissue. The surface of the body and even the mucous membrane of the mouth feel cold and stone-like. The bodily temperature is much reduced, falling often to 92° to 96° F. (33.3° to 38.6° C.). The respiration is slow and embarrassed. Circulation is poor.

**Pathology.**—The pathologic changes are in many cases obscure. In a case reported by J. W. Ballantyne there was found, on microscopic examination, to be an increase in the number and size of the connective-tissue bundles and an atrophy of the adipose tissue. Northrup reports a typical case in which no abnormal changes were found in the skin. Langer and others believe that solidification of the fatty tissues, in consequence of the very low temperature, is the cause of the hardening of the skin. It has also been suggested that in some cases the causes are very much the same as those producing myxedema. In many of these patients a postmortem examination will reveal an edema of the subcutaneous tissues, the secretion frequently being changed into jelly-like masses. Serous effusions into the pleura sometimes occur.

**Diagnosis.**—The only condition with which sclerema is likely to be confounded is that of general edema, from which it may be differentiated by the fact that in sclerema there is no pitting of the skin on pressure, by the rigid condition of the body, and by the great reduction in temperature.

**Prognosis.**—The prognosis is bad in nearly every case, the disease generally ending fatally in from one to four days.

**Treatment.**—The treatment consists of improving, as much as possible, the hygienic surroundings of the child, giving massage, galvanism, and also alcoholic stimulants in moderate doses. Such drugs as strychnin, camphor, musk, malt, and general tonics have been recommended. The administration of lanolin internally has been recommended. Thyroid extract has been said to be of service. When possible, the infant should be fed by a wet-nurse, or, if it cannot suck, the milk must be peptonized and given by gavage or the child may be fed by a medicine dropper inserted far back in the mouth. The bodily heat is to be maintained by keeping the child in an incubator at a fairly high temperature. In a case seen by one of us with Dr. Alexander Klein and which recovered, the treatment was prolonged baths in warm water, gentle massage and the administration of minute doses of antipyrine. We must admit, however, that this treatment was purely empyric.

#### MASTITIS IN THE NEW-BORN

It is a common occurrence to notice a secretion of milk in the breasts of the new-born infant—more frequently in male babies. The quantity of milk secreted varies from a few drops to probably a dram or more in exceptional cases. The milk secreted resembles colostrum, and by analysis has been shown to be composed of fat, sugar, proteids, salts, and water. It is often termed “witch’s milk.” The occurrence of this secretion is of very little importance, and usually ceases after ten or fifteen days, the secretion being most active from the eighth to the fifteenth day. The breasts show all the phenomena of physiologically active organs. Should the secretion be abundant, it can usually be dried up by painting the breasts with the tincture of belladonna. Strictly antiseptic washes with compresses may answer the same purpose. Undue pressure or slight trauma from careless or rough handling may cause the breasts to become inflamed, and thus set up a mastitis which may be more or less severe and even terminate in an abscess.

Mastitis is a rare condition in infants, but it is occasionally seen in a severe form and may lead to fatal results. The predisposing cause is the congestion which accompanies the active organ. The exciting cause is a trauma, micro-organisms gaining entrance to the breasts through abrasions or fissures produced by the trauma, or even, according to some authorities, through the milk-ducts. The affection is often due to a want of cleanliness. The symptoms are the usual ones seen

in inflammation of the breasts. Should the condition progress to supuration, extensive sloughing may take place, as shown in a case reported by Bush, although a single abscess is the more frequent result. The child is restless, there is a steady loss of weight, due to the fact that the child refuses to nurse, is peevish, fretful, and suffers from insomnia. The parts should be kept clean and no one allowed to press the breasts. A small cotton compress may be used for their protection.

In case acute inflammation develops, it should be treated as any inflammatory process. Hot antiseptic fomentations may be used in the beginning; should pus form, incise and drain. Keep up the child's strength with general stimulants and tonics.

### OBSTETRIC PARALYSIS

**Definition.**—A form of paralysis of the central or peripheral nerves occurring in the new-born and usually following prolonged efforts at forceps or manual extraction, or from other injuries to the head or extremities during birth.

**Etiology.**—The most frequent cause is attempts at using the forceps as a means of compression or forcible rotation to the fetal head instead of using the instrument as a tractor only. Occasionally, after delivery of the shoulder in a presentation by the breech or vertex, prolonged traction made by hooking the fingers in the axillæ will result in injury to the brachial plexus. One or more nerve-trunks may suffer traumatism, and from this will arise a form of paralysis of the arm known as Duchenne's paralysis. A very common form of peripheral paralysis is that known as "Erb's paralysis," or "the upper-arm type of paralysis." This arises from injury to the fifth and sixth cervical nerves. The muscles affected are the trapezius and, to a greater extent, the deltoid, biceps, brachialis anticus, supinator longus, or the supraspinatus or infraspinatus. Any or all of these muscles may be involved.

In a number of cases the violent separation of the head from the shoulders and the consequent stretching of the plexus at the junction of the fifth and sixth roots of the brachial plexus may be a cause, as has been pointed out by Walton, Carter, and others. The first-named author gives as his opinion that in most of these cases the plexus is, during labor, already brought against the clavicle, rotation of the head away from the affected side takes place, and at the same time the suprascapular nerve is put on the stretch between the point

of its emergence and the bony edge around which it passes to reach the infraspinous fossa. As the head separates from the shoulders after rotation has occurred, the shoulder being firmly held at the brim of the pelvis, its suprascapular nerve is stretched still further and the plexus bruised against the clavicle. As additional evidence in favor of his view the author calls attention to the fact that the right arm is generally affected in left occipito-anterior and right occipitoposterior positions and presentations, while paralysis of the left arm is most generally seen in those cases where the position and presentation have been right occipito-anterior.

When facial paralysis occurs, it is usually due to pressure by the forceps upon the seventh nerve at its point of exit from the stylomastoid foramen. This form of paralysis, although occasionally permanent, usually disappears in from a few days to a week or two after birth. A much graver and more lasting form of paralysis is apt to occur from injury to the brachial plexus in the manner before described. The pressure upon the cords of this plexus may either occur in the axillæ, or, as Ross has demonstrated, the fifth cranial nerve may be easily injured by a grip of the blade upon the upper arm and clavicle at a point where the nerve descends over the transverse processes of the fifth and sixth cervical vertebræ. Lane has also reported a case of injury to the brachial plexus by forceps—the face and arm were paralyzed. On postmortem examination a clot of blood was found at the stylomastoid foramen and around the cords of the brachial plexus.

Paralysis of central origin following labor may arise from pressure by blood-clot; thus, Lihotzky has reported cases in which pressure arose from this cause, probably induced by fracture of the orbital ridge by forceps. Hirst has reported a case of laceration of one of the sinuses of the dura mater caused by the overlapping of the parietal bones during labor. Precipitate labor, following which the child has fallen to the ground, has been given as a cause of paralysis in the newborn. Instances of cerebral atrophy with hemiplegia, either alone or associated with sensory and mental debility, and accompanied by frequent epileptiform convulsions, due in many cases to birth lesions, are reported by Allen Starr and others. Instances of the late effects of forceps compression have also been reported by Osler, who, in the records of the Philadelphia Infirmary for Nervous Diseases, found nine cases following instrumental delivery. Six of these had histories of direct injury by forceps, and some of them had marks on the head existing since birth. In all of these cases the paralysis appeared gradually a short time after birth.

**Symptoms.**—When paralysis of the arm or leg is present, there will be deficiency or absence of motion in the parts affected, and in a short time the muscles will appear soft and flabby, unless for a time masked by the abundant superficial fat. More or less complete anesthesia will be noticed. If the paralysis is of a mild type, recovery generally sets in early and may be complete. If severe, and the case remains in about the same condition for a long time, the chances for recovery are less favorable. As the peripheral nerves are not developed until several weeks after birth, any injury to them prevents their ever attaining functional competence. The response to electric tests varies with the degree and nature of the lesion; if it cannot be excited or if the reaction shows the anodal closure contraction to be greater than the cathodal contraction, then there is little hope. While the paralysis is usually confined to one nerve or one limb, it may be bilateral when the injury extends to both sides. If the face is involved, the first symptom generally noticed is a lack of movement of the facial muscles of one side; in some cases one eye may be injured, and drooping of the eyelid, contraction of the pupil, or retraction of the eyeball will be present; lack of expression on the injured side of the face and irregularity of the mouth will complete the picture.

**Diagnosis.**—Although paralysis due to causes incident to birth is generally easily diagnosticated, yet occasionally, from a medicolegal aspect, as well as that of the possible outcome of the case, it becomes necessary to consider certain points of differentiation. In cases of severe injury following the use of forceps, especially when compression has been used, a depressed spoon- or funnel-shaped mark will commonly be found on the areas of the fetal skull which have been within the grasp of the forceps. Occasionally these will be bounded by a well-defined ridge. The most common sites for these marks are the orbital and parietal regions and at the parieto-occipital junction. As a point of differentiation from the above, Fritsch has described the characteristic injury of the head received in falling after precipitate birth as follows: "The fracture begins in a suture and extends outward to the middle of the bone. Usually there is but one fissure, which ends where the bone is thickest. The parietal bone is the one most often injured, and the fissure usually ends in the parietal eminence." When the brachial plexus is injured, the paralysis affects but one arm, and will generally appear very soon after birth; usually while washing the child the nurse will observe that it moves but one arm, while the other hangs uselessly by its side. On examination, no evidences of a fracture of the bone will be found, and passive movement of the arm causes

little or no pain. The paralysis is not generally followed by contraction. Electricity will frequently be found to be of use in determining the injury: when the nerve is but slightly injured, there will be more or less response to the faradic current; but if the injury be great, there will be little or no response.

**Prognosis.**—The prognosis of facial palsy of simple form, and not the result of intracranial hemorrhage or fracture, is good. The muscles usually assume their activity at the end of a few weeks. When traumatism has occurred to the large nerve-trunks or laceration has resulted, or when the nerves have been injured to a large extent, the paralysis will be slow in disappearing and permanent injury may result.

**Treatment.**—The treatment of injuries of the nerves of the newborn may be divided into prophylactic and curative.

The prophylaxis requires the careful study of every case of delivery. By the accurate use of abdominal palpation and auscultation the position of the child *in utero* must be made out and the relative size of the fetus to the birth canal through which it must pass should be determined.

While the judicious use of the forceps to aid the expulsive forces of the uterus is fully justifiable, yet long-continued traction or the high application of the forceps without axis traction cannot be too strongly condemned. It is utterly useless and very dangerous to both mother and child to attempt to drag a fetus through a birth canal which is too small for it, and efforts to do this will be followed by nothing but disaster.

When, after a carefully made diagnosis, including the measurement of at least the conjugate of the pelvic inlet, it is found that the child is too large to pass, or, inversely, that the mother's parts are too small, operative procedures must be resorted to—either the Cesarean operation or symphysiotomy, and for the choice of these the student is referred to the text-books on obstetrics.

In cases where an infant is born with symptoms of cerebral compression following forceps delivery, the treatment by surgical means may be considered and the depressed bone elevated.

When injury to the brachial plexus has occurred, the treatment should consist of rest for the injured arm and, later, passive exercise. When the arm itself is injured, it should be wrapped in cotton batting and fixed to the side, care being taken that in bathing or dressing the child the arm is not allowed to hang down, as by so doing the injuries to the nerves may be increased. Tight bandages must be avoided.

At the end of four or five weeks the muscles may be treated by massage, shampooing, and galvanism.

As the progress of these cases is at best very slow, treatment must not be discontinued so long as any improvement, no matter how slight, continues. Massage and electricity do much good in these cases.

### UMBILICAL POLYPI

Polypoid granulations around the umbilicus occasionally appear and cause an oozing of blood. The treatment should consist of cleanliness and the application of some antiseptic, such as peroxid of hydrogen in a spray and solutions of silver nitrate.

The **prognosis** is generally favorable.

### DIVERTICULUM TUMOR AND PERSISTENCE OF THE OMPHALOMESENTERIC DUCT

Owing to the imperfect obliteration of the vitelline or omphalomesenteric duct during fetal life there may arise a number of abnormalities, among which are Meckel's diverticulum and a patulous condition of the omphalomesenteric duct. The former appears as a blind pouch on the convex surface of the lower part of the ileum, and may occasionally extend to the umbilicus. The omphalomesenteric duct exists as an open cylindric tube leading from the umbilicus to the ileum. (See *a, b*, Fig. 15.) Both the diverticulum and the duct are lined with mucous membrane similar to that of the ileum. When the diverticulum or duct connects with the umbilical opening, the mucous membrane of the duct becomes continuous with the skin at the navel and consequently there is noticed soon after the dropping of the umbilical cord a small reddish excrescence from which may escape mucus, occasionally bile-stained, and in some cases a small amount of fecal matter. Not infrequently the mucous membrane of the duct or diverticulum, and, indeed, in some cases the entire organ, may become prolapsed through the umbilicus, forming a tumor (see *c*, Fig. 15) which is known as a mucous polypus or diverticulum tumor. The tumor is usually small, about the size of a cherry or pea, and cylindric in shape and not pedunculated. It is covered with mucous membrane, and has at its most prominent part a small opening or fistula. By means of a probe or catheter this opening can be traced to the intestine. During straining at stool or paroxysms of coughing the entire duct may prolapse

through the umbilicus, and with it a portion of the proximal intestinal wall (see *d*, Fig. 15) or a small complete loop of intestine. (*e*, Fig. 15.) In these cases the tumor is larger, often the size of a man's fist, irreducible, is sausage-shaped and often bicornate. The covering of the tumor is the mucous membrane of the intestine. When a portion of the intestinal wall prolapses, there is but one external opening or fistula

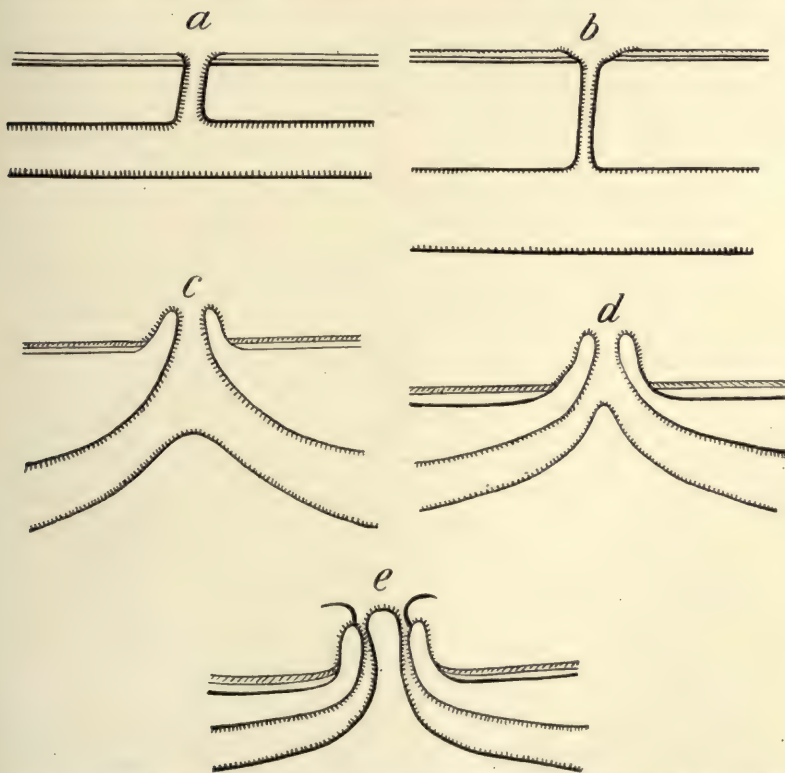


FIG. 15.—DIAGRAM ILLUSTRATING THE EFFECTS OF THE PERSISTENCE OF THE OMPHALOMESENTERIC DUCT AND THE FORMATION OF THE SO-CALLED DIVERTICULUM TUMOR.—(Riesman.)

which bifurcates a short distance from the surface, one portion going upward and the other downward. In case a complete section of the intestine has prolapsed, two openings can be seen, one opening passing into the upper and one into the lower segment of the bowel.

**Prognosis.**—When the tumor is composed only of the diverticulum and not accompanied by serious prolapse, the prognosis is good. Prolapse of the duct alone is always serious, and when accompanied by the bowel, the result is usually fatal (Riesman). One of the dangers of the condition arises from the fact that these tumors are often mis-

taken for other growths, and operative procedures are attempted which are followed by disaster. Strangulation of the bowel, caused by the diverticulum or duct, may also occur.

### UMBILICAL HERNIA

Hernia through the umbilical ring arises from imperfect closure of the parts. The congenital form may be the result of the non-closure of the ventral lamina, or it may be due to the continuance of the fetal condition, in which a coil of intestines remains outside the abdominal cavity, the result of imperfect closure of the anterior walls of the latter. In other words, there is an arrest of development of the abdominal walls, while one or more coils of intestines, which, during embryonic life are developed outside the abdominal cavity, fail, by the deficient development of the latter, to be inclosed in the usual way. Climate seems to have some effect as a cause of umbilical hernia; thus, according to Wert, Spain and Portugal have the highest percentage of cases of hernia in proportion to the population, and South America the lowest. In the United States, Minnesota has the highest average and West Virginia the lowest. Umbilical hernia may be congenital or acquired. In the congenital form the most frequent cause is the arrest of development of the abdominal walls, as before described. Another cause of this variety of hernia is probably the failure of the normal process of atrophy of the umbilical vesicle. The acquired form is much more generally seen in badly nourished, poorly developed children, or in those who have been weakened or debilitated by disease. It is not infrequently seen in children with chronic diarrhea or in the same class of children who, from any reason, have violent attacks of vomiting or coughing. An elongated uvula may act as a secondary cause of hernia by producing violent efforts at vomiting or coughing. Rectal polypus has also been given as a cause. No matter from what source the hernia arises, it will appear as a soft tumor in the center of the umbilical ring. This tumor will be increased by the act of coughing or efforts of bearing down. The site of the protrusion, while usually occurring in the position named, occasionally appears above the ring between the recti muscles. In this latter instance it is more properly called ventral hernia.

**Treatment.**—In early life a complete cure can often be effected by drawing the two sides of the ring together after replacing the hernia, and passing bands of rubber adhesive plaster about the abdomen so as to keep the ring closed. The strips should be about  $1\frac{1}{2}$  of an inch

wide. This dressing must be repeated from time to time until the intestine ceases to protrude. Care must be taken, however, that the delicate skin is not chafed by the adhesive plaster. To prevent this the skin may be dusted with some mild aseptic powder before dressing.

The ordinary treatment consists of covering a convex button with cotton or buckskin and making pressure against the ring with the convex side of the button, the latter being held in place by a bandage around the abdomen.

Operative treatment is sometimes useful. When the umbilical ring remains open and surgical means cannot be tried, the hernia must be replaced and the opening closed by a suitable truss. This truss usually consists of a convex rubber button held in place by a spring or a rubber bandage.

### OPHTHALMIA IN THE NEW-BORN

By this term is usually meant a form of conjunctivitis occurring in new-born infants, produced in the majority of cases by infection from the vaginal secretion of the mother, who has previously been infected with gonorrhea. Ophthalmia may also be caused by pneumococcic infection and in some rare instances by the streptococcus and staphylococcus. In by far the largest number of cases it is caused by the gonococcus. In mild or moderately severe cases the disease is limited to the eyelids, the conjunctiva, or the subconjunctival tissues, but when the infection is severe, there may be ulceration of the cornea or even perforation of the eyeball.

**Etiology.**—Mild cases of inflammation and swelling of the eyes may be produced by continued pressure during birth, or from the eyes of the child coming in contact with a vaginal discharge if the mother suffers from a simple catarrhal vaginitis. Even a healthy lochial discharge may produce a slight inflammation. True ophthalmia is, however, in almost all instances produced by the eyes of the child coming in direct contact, during labor, with a vaginal discharge which is either infected with the gonococcus or with ordinary pyogenic germs.

Uncleanliness on the part of the nurse—as, for instance, in the dressing and care of an infant after attending the mother who has been infected—may be a cause.

**Symptoms.**—The disease usually begins about the third day after birth, the first symptoms being redness and swelling of the lids, the conjunctiva, and the subconjunctival tissues, and is immediately followed by a very free purulent discharge. The discharge is yellowish

or greenish in color and may be blood-streaked; sometimes, indeed, there may be distinct hemorrhages. In a short period of time ulceration of the cornea, followed by sloughing, takes place, and unless the disease yields to treatment, a perforation into the anterior chamber of the eye follows. When gonorrheal poisoning has been the cause, loss of vision very often results unless the case is seen early and very energetic and careful treatment is instituted. Neglected cases may end not only in loss of sight, but in systemic poisoning by pyemia.

**Treatment.**—The prophylaxis of ophthalmia in the new-born consists in first giving every woman affected with any form of purulent vaginitis or endometritis an antiseptic douche during the first stage of labor. The vagina should be thoroughly flushed with a 1:2000 or even 1:1000 solution of bichlorid of mercury, or a hot solution of creolin and tincture of green soap, equal parts, in the strength of one dram to the quart. This should be given through a speculum, care being taken that all the folds of the vagina be stretched and the whole birth canal thoroughly swabbed by means of wads of cotton held in a pair of uterine dressing forceps. The next step in the preventive treatment consists in dropping into the infant's eyes, immediately after birth, a 1 per cent. solution of nitrate of silver, or a 10 per cent. solution of argyrol or protargol. If nitrate of silver is used it should be followed by a drop or two of salt solution in order to neutralize any excess of the nitrate of silver. In some cases a thorough cleansing of the eye with a saturated solution of boric acid does well. When the eye has become infected, the treatment must be prompt and each step accurately carried out. The strictest antiseptic precautions must be used to prevent the infection from spreading to the other eye where only one is affected, and also to keep the disease from attacking those who attend the patient, or, in hospitals, from spreading to other inmates. All cases of ophthalmia neonatorum should be isolated, and a special nurse be set apart to attend such patients.

The treatment consists first in cleansing the eye every twenty or thirty minutes, night and day. This should be done by instilling into the conjunctival sac solutions of boric acid, by means of the ordinary grooved eye-dropper or a dropper with a bulbous tip. Care should be taken that the fluid is introduced well into both angles of the eye, and without force, taking care to empty the region thoroughly of pus. In order to reduce the inflammation there should be constantly applied to the eye small pieces of lint or absorbent cotton—compresses, in fact—which have been rendered cold by letting them lie on a cake of ice. These should be applied every minute, at least, and this treat-

ment should be continued as long as any discharge continues, taking care that these are not used should the cornea in any way become involved (haziness, ulceration, etc.). Such cases are then much better treated by the substitution of heat until the cornea returns to its proper condition, when cold may again be used. It has been recommended that a few drops of a 1 per cent. solution of nitrate of silver should be instilled into the eye. In all cases the pupils should be dilated with atropin. It is of the utmost importance that all dressings, etc., coming from the patient should be burned as soon as removed, and the nurse must take the greatest possible care in washing her hands both before and after attending the case.

## CHAPTER III

### GENERAL HYGIENE OF INFANTS AND CHILDREN

As soon as the child is born it should be laid on its side upon the bed, far enough away from the mother to prevent her rolling upon it. The first attention after severance of the cord should be directed to the breathing, care being taken that inspiration and expiration are regular. Any accumulation of mucous in the mouth should be removed by a finger covered with soft muslin or lint soaked in a solution of boric acid. Should the respiration be weak, the child should be held head downward and a small stream of cold water poured on the chest. Slapping the buttocks or chest with a towel wrung out in cold water will often aid in the establishment of respiration. When asphyxiation occurs, it should be treated according to methods previously described. Care must be taken to see that no bleeding occurs from the umbilical stump, and that the ligature is fastened securely. The material used for the ligature must be sufficiently strong to hold without slipping, and at the same time not be so thin as to cut through the jelly of Wharton or possibly the umbilical vessels. Frequently if slipping of a ligature does occur in from twelve to twenty-four hours after birth, the vessels are sufficiently contracted to prevent hemorrhage; indeed, we have observed in at least two cases in which the ligature has slipped but a few hours after birth that the hemorrhage has proved very slight. The material used in tying the cord should be a double strand of linen or thread. A stout ligature of twisted silk will do very well. The ligatures in all cases must be rendered aseptic before applying.

The eyes of the new-born infant are one of the most important objects for attention. As soon as respiration is established and the cord tied, the eyes should be carefully washed with a solution of boric acid; in many cases this is sufficient. However, it is generally recommended that a drop or two of a solution of five grains of nitrate of silver or a 10 per cent. solution of argyrol to the ounce of distilled water be dropped in the eyes from a medicine dropper. When a solution of silver nitrate has been used, this may again be advantageously followed by carefully mopping the eyes with sterilized water after any excess of silver nitrate has been neutralized by instilling a few drops of a .6 per cent. solution of sodium chlorid. When the mother has had a puru-

lent vaginal discharge, it is often necessary to instil into each eye a few drops of a 1: 12,000 solution of bichlorid of mercury, followed by a similar application of distilled water. Before giving the infant its first bath it is necessary, on account of the sticky, cheese-like secretion which covers the skin (*vernix caseosa*), to smear the child's body with either olive oil or vaselin. Unless this is done, the *vernix caseosa* is extremely difficult to remove. The first bath should be given in a room the temperature of which is from 65° to 70° F. (18.3° to 21.1° C.); the temperature of the water should not exceed 96° F. (35.6° C.), as the skin of the new-born is extremely sensitive, and a too hot bath may cause irritation thereof, which may amount to an actual dermatitis. Soap may be used in this bath, providing it is free from strong alkalies, the best soap being a superior grade of Castile, or, as some recommend, Unna's "overfatty" soap. After bathing, the skin should be mopped dry with a soft towel, after which it should be dusted with a powder consisting of 2 per cent. of salicylic acid and finely powdered starch, or boric acid or thymol, 5 per cent. in starch powder. Some prefer to use the oil alone, wiping this carefully off after applying, and to use no water for several days or weeks.

The infant should be dressed in a soft, unstarched material, made loose, so as to prevent pressure and allow perfect freedom of motion. It is well to avoid all excessive ornamentation with lace. The diapers should be made of soft absorbent material, and a sufficient number provided to allow of change and washing after each evacuation of the bowels and bladder.

The cord-stump should be dusted with any of the antiseptic powders before referred to, and may be laid so that the severed portion points upward, and covered with a little bag of gauze or a small pad of absorbent cotton, held in place by an abdominal binder of light flannel, care being taken that this bandage is loose enough not to interfere with respiration.

As soon as the child is washed and dressed it should be laid in a small crib by itself. It should never be allowed to sleep in the same bed with the mother, as there is danger of her rolling over on it during sleep and causing suffocation.

Shortly after the child is washed and dressed it should receive a teaspoonful or two of hot sterilized water; this acts on the kidneys, aiding in the establishment of urination and stimulating renal action generally. It is well to put the child to the breast from two to four hours after birth, or as soon as the mother is adequately rested.

From birth to the end of the sixth or eighth month the infant should

sleep from 11 P.M. until 5 A.M., and as many hours during the day as nature demands and the times of feeding, washing, and dressing will permit. From the eighth month to the end of two and a half years the child should sleep from noon until 1.30 or 2 P.M., and at the time of taking this nap it is to be undressed and put to bed. At 7 P.M. it should be put to bed for the night. When the child reaches the age of from two and a half to four years, the morning nap may occasionally be omitted, according to indications, but in all cases the time for the night's rest properly begins at 7.30 P.M., and should last until 6 or 7 A.M. After the fourth year the daytime nap need not be insisted upon, but the child should be put to bed by 8 P.M., and sleep for at least ten hours. When possible, the sleeping-room and the room occupied in the day ought not to be the same; or, when this is not feasible, the child should be removed to some other room for an hour or two before retiring for the night and the sleeping-room well aired. The temperature of the sleeping-room should be from 64° to 68° F. (17.8° to 20° C.), and this temperature is to be maintained as uniformly as possible.

**The Bath.**—The child should be bathed at a regular time each day, one bath in the twenty-four hours being considered enough. This should be given during the morning, at a time about halfway between the first two feedings. The temperature of the room should then be about 70° F. (21.1° C.), and kept free from drafts. The first step in bathing should be to wet the child's head thoroughly in order to prevent its taking cold. The duration of the bath should be from three to five minutes, after which the skin must be well dried with a moderately coarse towel. After the bath the body should be covered with a blanket or flannel night-robe, and the infant put to sleep again for a short time. It is strongly recommended that occasionally in hot weather an additional sponge-bath of water at a temperature of 90° F. (32.2° C.) be given, which will have a cooling effect upon the skin. In older children cool baths at a temperature of from 72° to 76° F. (22.2° to 24.4° C.) are sometimes more valuable than warm ones. Very cold baths, except in rare conditions, are not to be recommended for children. They are, however, occasionally useful as a tonic or stimulant, increasing the excretive powers of the skin and giving tone to the body. These baths must always be given in a warm room, the child standing in enough hot water to cover the feet. The cold water should be applied by means of a sponge, one sponging of the whole body being sufficient. The temperature of the water used ought not to be below 64° F. (17.8° C.). In many cases the addition of an ounce or two of sea-salt or ordinary rock-salt will increase the good effects of the bath,

which should be followed by a thorough rubbing with the hands and a coarse towel. Another method of giving a cold bath is to allow the child to stand in hot water while the body is enveloped in a sheet wrung out of water at a temperature of 60° F. (15.6° C.), and the entire surface of the body well rubbed through the sheet, after which the child is rubbed with a towel until the skin is thoroughly dry. This method is applicable to older children.

A bath at a temperature of 94° to 100° F. (34.4° to 37.8° C.) is frequently used to produce diaphoresis, to relieve nervous irritability, and to promote sleep. When considerable stimulation is required, mustard may be added to the water in quantities from a teaspoonful to a tablespoonful. As a general rule, five minutes is long enough for immersion in a hot bath.

**Exercise.**—Muscular exercise in some form is necessary to the maintenance of health. Nature provides this in young infants in the frequent motion of the whole body, so that all that is necessary is to undress a young baby and let it lie on its back and kick and move at will. As the child begins to creep, and later to walk, the muscles of locomotion and of coordinated action are slowly developed.

A baby may be taken out-of-doors in from three weeks to a month after birth, and from that time on it should be kept in the open air for a certain part of every day, providing the weather permits. Moderately cold weather, if the air is dry and there is no wind, need not keep any but a very young infant indoors; the child, however, should be well wrapped up. In hot weather the head should be protected, and the child should, of course, be kept away from the direct rays of the sun. Exercise in older children is best managed in the moderate use of the ordinary games, especially those which take the child out-of-doors. Games not only help to develop the muscular system, but also give the child an object to obtain in mastering them; besides this, the obedience taught by the rules of games affords a certain discipline which acts for their good. In stormy weather children may be warmly clad and allowed to run about and play in a room with the windows open. As a rule, it is best not to allow a child out-of-doors at night.

**Care of the Mouth.**—The mouth of the newly born infant should be gently cleansed every day with boiled water and a soft cloth. The hands of the mother or nurse should be clean before attempting to wash the mouth of the infant. Too frequent or any but the most gentle methods of doing this are to be discouraged, as the epithelium of the mouth of the infant is very delicate and much harm may be done by injuring it. From the appearance of the first tooth on through to

the cutting of both temporary and permanent sets, the teeth should be carefully and gently brushed once or twice a day. Neglect of this predisposes the teeth to become carious, and should this occur, the child should be referred to a good dentist and have the carious teeth filled or extracted. Carious teeth are not only unsightly, but they give rise to bad breath, pain, and frequently form the starting-point for infection.

**Care of the Genital Organs.**—The genital organs of children should receive attention, particularly as to cleanliness. In the male child if the foreskin is long and the preputial orifice of normal size, simply drawing back the former and carefully, but thoroughly, cleansing the glans with warm borax water or with Castile soap and water is enough. When the preputial orifice is somewhat contracted and adherent, the adhesions should be broken up by gently rotating the closed blades of a small pair of dressing forceps about the glans, after which the prepuce should be stretched gradually from day to day until the foreskin can be drawn easily back over the glans. When the foreskin is very long, circumcision should be done though, unless performed as a sacerdotal rite, it is best postponed until a year has passed. We have known of three deaths in one family from hemorrhages and also of infections that proved fatal in several instances.

The genital organs of female children require little but simple cleanliness.

**The Nursery.**—The nursery must be a fairly large room, preferably facing the south, so that plenty of sunlight can enter it freely. If possible, it should not be on the ground floor or, on the other hand, it should not be at the top of so many flights of stairs as to be inconvenient. It should be heated by an open fire or, as Holt recommends, by a Franklin radiator. Steam heat or gas should not be used. Ventilation is of the greatest importance, and at the same time direct drafts are to be avoided; this can be accomplished best by any of the usual forms of ventilators which are designed to be placed in the windows. The furniture should be simple in style, not too much in quantity, and of plain, solid surface, not basket woven, so that it can be easily cleaned. All heavy hangings are to be condemned. The floors should be covered with rugs, tightly fastened to prevent the child or nurse with the baby in her arms from tripping. The bed on which the child lies should be furnished with a hair mattress and a pillow of the same material; no hangings of any sort should be used about the bed. Cradles that rock are an abomination, and should be excluded. For lighting a nursery oil or gas or shaded electric lamps may be used; the former

presents many points of usefulness providing the lamps can be so placed that they cannot be upset; on the whole, gas or electricity are to be preferred. At night a small wax night-light is all that is required. The temperature of the room should not be above  $70^{\circ}$  F. ( $21.1^{\circ}$  C.) during the day and about  $64^{\circ}$  F. ( $7.8^{\circ}$  C.) at night. The nursery should not be used as a place for drying diapers and clothes generally; nothing can be more unhealthy, not to say disgusting, than to see a line filled with diapers hanging in front of a nursery fire—a sight much too familiar to many physicians.

## CHAPTER IV

### FEEDING AND FOOD OF INFANTS AND CHILDREN NUTRITION AND THE IMPORTANCE OF PROPER FOOD IN EARLY LIFE

Of all the problems of our physical existence probably none is more important than nutrition. To go a step further, it is but a truism to state that the mind has its best opportunity for a normal development in a well-nourished body. Happiness itself may depend largely upon proper nutrition. Unhappiness, crime, and vice, on the other hand, may directly or indirectly depend upon malnutrition, or the factors that produce it (starvation, alcoholism, etc.). Now when we view the enormous advances in weight and height which the human body undergoes in the period of infancy (see Chapter I), when we note in particular the relatively rapid growth of the brain, it seems but logical to admit that problems of nutrition are more important in infancy than at any other periods of life. And, having advanced this far, another obvious truth must stare the conscientious medical man in the face: if he treats children at all, it becomes his manifest duty to see that they are properly nourished.

The word nutrition, in its broadest sense, implies a great deal. Proper food rests at its very base. The processes of digestion next engage our attention. The term absorption implies processes of vast importance in nutrition. Tissue metabolism—the “sum total of the chemico-vital processes” in the tissues—must be studied in its important rôles of anabolism and catabolism. Secretions and excretions are other important phases of nutrition of which we have learned much and shall learn much more in the near future. Nor must we forget, in a broad consideration of nutrition, the important problems of activity and rest; of the amounts of energy expended in heat, muscle activity, digestion, growth processes, mental operations, etc. But, as we have remarked, at the very basis of nutrition, we find food.

#### MATERNAL FEEDING.—HUMAN MILK THE PROPER FOOD FOR THE INFANT

Human milk is a secretion of woman's mammary glands, representing a solution of sugar and salts, of proteids partially in solution

and partially in suspension, in an emulsion of fats. Its color varies from bluish-white to yellow, and, according to most authorities, is no index of its richness. Normally its specific gravity ranges between 1029 and 1032 (Holt). Exceptionally specific gravity may vary between 1026 (very poor) and 1036 (very rich). As it emerges from the ducts of the mammary glands human milk is practically sterile.

Our conceptions of the chemical composition of mother's milk are rendered much more clear if we compare it constituent for constituent to the blood or lymph from which its elements are drawn. In the blood or lymph, we find inorganic and organic constituents; and so we do in milk. Both circulating fluids contain water and salts (or ash); so does the secretion of the mammary glands. The proteids or proteins are comparable; for blood has its fibrinogen, its serum albumin and its serum globulin; milk its caseinogen, lactalbumin and lactoglobulin. Carbohydrates are represented in all three fluids, blood and lymph containing dextrose; while the sugar of the milk is lactose. Fats (hydrocarbons) are also found in blood, lymph and milk. It is not to be supposed that this is other than a qualitative comparison; for the percentages of these various constituents differ considerably in the fluids mentioned. Indeed, the term secretion, as we understand it, implies of necessity, differences in both quality and quantity between the product of glandular activity (milk) and the circulating fluids. "Substances are selected from the lymph by the glandular cells and contributed in an altered (organic secretion) or unaltered (inorganic secretion) state for the further use of the economy. Milk differs from other secretions of the body, in that the inorganic and organic substances are contributed for the use of another organism.

The following table, exhibiting as it does analyses of prominent authorities in various lands, yields very valuable information concerning the composition of human milk. (From Judson and Gittings.)

	Pfeiffer	Leeds	Johan- nessen	Rich- mond	Leh- mann	Meigs	Schloss- mann	Adriance
Number of cases	160	80	25	90	40	43	218	120
	%	%	%	%	%	%	%	%
Fat.....	3.11	4.13	3.21	3.07	3.8	4.28	4.83	3.83
Lactose.....	6.3	6.93	4.67	6.59	6.0	7.4	6.95	6.56
Proteins.....	1.94	1.99	1.10	1.97	1.7	1.05	1.05	1.30
Salts.....	0.19	0.20	.....	0.26	0.20	0.10	.....	0.20
Water.....	88.22	88.73	.....	88.04	88.5	87.16	.....	87.80
Solids.....	11.76	13.26	.....	11.89	11.70	12.83	.....	12.20

Droop Richmond, compiling his table from the first five of these analyses, considering them most reliable, says that the following may be accepted as the average composition of human milk:

Water.....	88.2 per cent.
Ash.....	.2 per cent.
Proteids.....	1.5 per cent.
Fat.....	3.3 per cent.
Sugar.....	6.8 per cent.

Probably the figures of Holt are more closely in accord with the results of modern analyses (Pfeiffer, Koenig, Leeds, Harrington, Adriance, etc.).

	Normal average	Common healthy variations
Fat.....	4.00 per cent.	3.00 - 5.00 per cent.
Sugar.....	7.00 per cent.	6.00 - 7.00 per cent.
Proteids.....	1.50 per cent.	1.00 - 2.25 per cent.
Salts.....	.20 per cent.	0.18 - 0.25 per cent.
Water.....	87.30 per cent.	89.82-85.50 per cent.

The fact must again be emphasized that these figures only represent averages. Milks of different mothers may differ considerably and yet their respective babies may thrive quite normally (Rotch, etc.). Again, the milk of the same mother may vary at different times of the day and under varying conditions of diet, exercise, etc., and yet her baby may do splendidly. It has been suggested that such variations may actually be of advantage to the baby. They probably are.

It will now be well to discuss at greater length the various constituents of human milk:

**The Proteids.**—As already stated the principal proteids are casein (really caseinogen), lactalbumin and lactoglobulin. Opalisin, a proteid rendering its solutions opalescent, has also been detected in human milk. The proportion of casein (non-coagulable proteid) to other proteids (coagulable by heat) has been a subject of discussion for a number of years. Casein in human milk exists in larger amounts than was formerly thought. Raudnitz (Pfaundler and Schlossmann) gives

Casein.....	0.6-1 per cent.
Lactalbumin and lactoglobulin.....	.5 per cent.

The figures quoted by Fischer and others are closely in accord:

Casein.....	1.2-1.03 per cent.
Albumin.....	.5 per cent.

Nevertheless, as has been known for many years, the casein of mother's milk in the presence of serum or acids coagulates in much finer flakes than the casein of the milk of ruminants. It contains more phosphorous than cows milk. The principal point that we wish to make is that the casein of mother's milk differs from the casein found in the milk of cows or other animals.

Lactalbumin from a given milk possesses the same properties as the serum albumin of the animal's blood. As we shall soon find, it and lacto-globulin are found in large amounts in colostrum. Woman's milk is said to contain twice as much nuclein as cow's milk. It also contains more lecithin, which because of its possible union with proteids (lecith-albumin) we shall mention under this heading. Indeed, all of the phosphorous of human milk is thought to be in organic combination with casein, nuclein and lecithin. Less than half of it is in organic combination in cow's milk.

Lastly it is worthy of note that the proteids of milk are specific; "that is, their injection into rabbits occasions in the serum of the blood the formation of a substance which causes a precipitate with the milk or the proteids of the same, or a closely related species of animals." (Raudnitz, "Pfaundler and Schlossmann").

**The Fats.**—The fats are glycerides of their corresponding fatty acids. It is stated that as many as fourteen fatty acids may be obtained from milk fat. Judson and Gittings mention butyric, caproic, myristic, palmitic, stearic, and oleic acids. "According to Ruppel, mother's milk is comparatively poor in *volatile* fatty acids. Of the non-volatile, oleic acid forms one-half. Palmitic and myristic are in excess over stearic acid." Raudnitz's figures show that the milk of the cow contains approximately from four to six times as much volatile fatty acid as human milk. This last statement must be pregnant with suggestion to every pediatrician; but the point that we would emphasize for the student is that the fats of mother's milk, like the proteids, differ essentially from the hydrocarbons found in the milk of other species. Like the nitrogenous elements, they are specific.

We are inclined to agree with authorities who emphasize the value of the microscopic study of fat globules. All are agreed that we find (1) large, (2) medium, (3) small fat globules. The second group should preponderate numerically in good mother's milk. The approximate number of globules should also be noted or they may be actually counted as we count red blood cells. (See analysis of milk.) During the progress of lactation, the fats vary more than any other constituent of the

milk; but any percentage below 2 per cent. or above 5 per cent. may be considered abnormal (Judson and Gittings).

**Milk Sugar.**—Most authorities agree that lactose as found in mother's milk is identical with the milk sugar of other species.

**Salts.**—We shall compare here the salts of human and cow's milk, using the comparative table given by Judson and Gittings:

MOTHER'S MILK. (Harrington and Kinnicutt.)	Cow's MILK. (Adapted from Söldner.)
Sodium chloride..... 21.77	Sodium chloride..... 10.62
Potassium chloride..... 21.05	Potassium chloride..... 9.16
Potassium sulphate..... 8.33	Potassium citrate..... 5.47
Potassium carbonate..... 23.47	Potassium phosphate..... 21.99
Calcium phosphate..... 23.87	Calcium phosphate..... 16.32
Calcium carbonate..... 2.85	Calcium citrate..... 23.55
Calcium sulphate..... 2.25	Lime combined with proteids..... 5.13
Calcium silicate..... 1.27	Magnesium citrate..... 4.05
Magnesium carbonate..... 3.77	Magnesium phosphate..... 3.71
Iron oxide and albumin..... 0.37	

It is more than probable that the differences between the salts of human and bovine milk have not been sufficiently appreciated in the past, and that we shall pay much more attention to them in the future than we do now. A mere glimpse at the table will serve to reveal the wisdom of Jacoby's use of salt (sodium chloride) in his milk mixtures. It will also furnish an *a priori* argument against the routine use of lime water in modifications of cow's milk. Some of the lime in cow's milk is in combination with casein (calcium-casein) and aside from the fact that cow's milk already contains more calcium salts than mother's milk, the addition of more alkali may only serve to delay the action of rennin in the presence of hydrochloric acid. The views just enunciated are probably of considerable practical value, but we would subvert them all to have the student again grasp the fundamental and essential fact that these constituents differ from the salts of other milks.

The salt content of mother's milk varies usually but little from .2 per cent. It is about one-fourth of the amount in cow's milk.

**Other Constituents.**—In addition to the substances we have just studied in some detail milk contains gases, "citric acid, substances producing colors and odors, ferments and alexins."

It might be of considerable interest to study the various ferments of milk; but we feel that the student should be referred to more exhaustive treatises for such information. For our purpose, suffice it to say that some ferments are found in the milk of some species and not

in others; and that they also vary qualitatively in the milks of different species. Another point of great interest is that the ferments of cow's milk begin to be destroyed by temperatures above  $140^{\circ}$  F. (Rosenau, Freeman, etc.).

The alexins (complements) of milk are derived in part from the transmitted serum and in part from the glandular cells (Raudnitz-Pfaundler and Schlossmann). "All the alexins produced in the body of the (mother) animal, whether by experiment or disease, pass into the milk." How much light is thrown upon the question of infantile immunities to many diseases by a moment's consideration of this statement?

**Colostrum.**—During the latter months of pregnancy a few drops of this substance can usually be expressed from the prospective mother's breasts. In the first two days of the baby's extra-uterine existence, the amount of this fluid steadily increases. On the third day after birth, the milk usually "comes in" and gradually replaces the provisional fluid, colostrum.

Modern conceptions favor the view that colostrum possesses the qualities of blood rather than the qualities of milk; that the colostrum period represents for the infant a transition stage between placental nourishment and breast (milk) feeding.

Practically colostrum contains more salts than does the later milk. It may contain more or less fat (probably the former is the rule). Its sugar content is nearly always lower than that of milk. In the cow it is dextrose. The proteid content is usually higher than in milk and lactalbumin and lactoglobulin (analogues of the serum proteids) are in excess of the casein.

Colostrum characteristics should have disappeared from the milk in approximately two weeks' time.

Fischer quotes five analyses of human colostrum made by Harrington:

	Case 1	Case 2	Case 3	Case 4	Case 5
Fat.....	1.40	0.68	2.40	5.73	4.40
Lactive and proteids.....	9.44	11.53	11.15	10.69	11.27
Ash.....	.17	.31	0.25	0.16	0.21
Total solids.....	11.01	12.52	13.80	16.58	15.88
Water.....	88.99	87.48	86.20	83.42	84.12

"Microscopically there are found: (a) fat droplets, some like those of normal milk and some smaller, poorly formed and often agglutinated, which denote the imperfection of the milk secretion; (b) Leukocytes, some of which contain fatty detritus; (c) colostrum corpuscles, large spherical bodies, consisting of fatty detritus surrounded by a membranous envelope and often showing ameboid movements" (Judson and Gittings). A. E. Taylor, on the other hand, found that most of the granules in colostrum corpuscles showed the reactions of proteids and not of fats.

The functions of colostrum are well stated by Chapin:

1. To cause a discharge of meconium, the contents of the fetal intestines, so that food may pass and be absorbed.
2. By the large proportion of easily absorbed forms of protein and sugar it contains, to start up the absorption process and nourish the infant.
3. By the gradual substitution of protein and sugar that need digestion to stimulate the flow of the digestive juices.

Again we would emphasize the obvious fact that colostrum presents characters midway between blood and milk, and that the colostrum period represents an intermediate stage of nourishment.

We quite agree with Jacoby that the colostrum period may be a disturbing or even dangerous one to the infant, and shall deal with this matter under another heading.

### THE ANALYSIS OF MOTHER'S MILK

The individual baby is usually the best laboratory for the individual milk. If he is physically comfortable, gains in weight consistently and in proper degree, the mother's milk that he is taking is suited to his individual requirements, even though analysis makes its chemical composition seem bizarre. If, on the other hand, he has gastric or intestinal colic, fails to gain in weight or actually loses, if he vomits unduly or his stools are abnormal, the milk that he is receiving, no matter how ideal theoretically, is not suited to his particular needs.

So we shall begin the question of analysis by making some practical criticisms, hoping thus to place the whole matter in a fairer and more lucid light:

1. A mother's milk may vary considerably in composition from time to time, and its composition depart decidedly from average standards, and yet it may agree perfectly with her baby.
2. A seemingly ideal milk, chemically, may disturb another baby. This fact is of practical importance in wet-nursing.

3. Unless the whole breast is emptied for purposes of analysis, or unless samples are taken from the mid-milking, chemical studies may produce very faulty results. Thus we know that the fore-milk is poor in fat and that the last milk ("strippings") is very rich in fat.

But after all has been said in criticism, there still remains a considerable field of usefulness for chemical analyses of breast milk. Such an analysis often throws light upon the causes of infantile disturbances. It may show us that a milk is hopelessly deficient in solid constituents. It may prevent us, on the other hand, from absolutely abandoning breast feeding. We feel that such analyses are not made nearly often enough.

**An Illustrative Case.**—One of us very recently secured 1 and  $1\frac{1}{2}$  ounces of milk from the breast of a mother who thought her supply deficient. Her baby weighed but 5 pounds and 6 ounces, so that in one breast there was more than enough for a single nursing. Analysis showed 3.6 per cent. fat (Babcock), and a specific gravity of 1029.8. Applying the rule of Farrington and Woll (see below), the total solids equalled 11.8 per cent. There was really nothing to show that the baby was disturbed by his food. He was suffering from intense jaundice and from a large right inguinal hernia.

**Securing the Specimen of Milk.**—A method suggested by Klotz has served us very well. A nursing bottle (or better, a larger, wide-mouthed milk bottle) is boiled. Cool sterile water is then permitted to run over the mouth of the bottle. The mouth is then immediately placed upon the breast, so that the nipple is enclosed by it. As the air in the bottle cools, the breast milk will flow steadily into it. We complete the emptying process by massage of the breast. This method we have found more efficient than the use of a breast pump. More than this, the milk is not only ready for analysis, but having been drawn into a sterile container, it is fit for use as an infant food, if it is needed for that purpose.

We then follow the method so clearly outlined by Chapin: 1. Calculate the fats by the means of the Babcock or some other centrifugal apparatus. 2. Take the specific gravity with the Quevenne lactometer or a smaller hydrometer. 3. Calculate the solids not fats by the rule of Farrington and Woll.

1. **The Calculation of Fats.**—If there is enough milk in the breast to nourish an average infant, we may secure enough to make this study. If the amount is small, we study the specific gravity first, and then use the milk in the centrifugal apparatus.

With a pipette we draw up 17.6 c.c. of milk. This is placed in

one flask. We then keep a similar amount in a second flask. To each is then added 17.5 c.c. of commercial sulphuric acid. Successively the flasks are then gently agitated. The sulphuric acid chars all of the solid constituents except the fats. Considerable heat is generated and the fats rise to the top. The flasks are then placed in the centrifuge and are whirled for five minutes. Boiling water is then carefully added to each flask until the fat layer comes well up in the neck of each; the bottles (flasks) are then whirled for another minute. Boiling water is then poured over the neck of each flask, and the bottles are whirled for another minute. The percentage is read off directly, reading from the top of one meniscus to the bottom of the other. Thus in a recent test the upper meniscus was at 7.6, the lower at 4. Now 7.6 per cent. - 4.00 per cent. = 3.6 per cent. of fat.

2. **The Specific Gravity.**—We like the Quevenne lactometer, because it contains a thermometer, and corrections for temperature may be immediately made. One may read off the specific gravity directly instead of transmuting figures, as with the "Board of Health lactometer" or the creamometer.

3. **The Solids not Fat.**—These are estimated by the rule so carefully worked out by Farrington and Woll: To one-fifth of the fat percentage add one-fourth of the last two figures of the Quevenne lactometer's reading. The result is the percentage of solids not fat. Add to this the percentage of fat, and we have the total solid percentage. Thus in the example given above, the percentage of fat was 3.6 per cent.; the Quevenne lactometer reading 1029.8. —

$$\frac{3.6\%}{1} \times \frac{1}{5} = .7$$

$$\frac{29.8}{1} \times \frac{1}{4} = 7.5$$

Solids not fat = 8.2

Adding the fat percentage = 3.6

We have the total solids = 11.8

Further than this: If we assume the sugar content to be 6.8 per cent., and the salt content to be .2 per cent., and both are pretty constant, we may roughly estimate the percentage of proteid constituents:

Solids not fat = 8.2 per cent.

Sugar and salt percentages (6.8 + .2) = 7.0 per cent.

Estimated proteid percentage = 1.2 per cent.

Very convenient for a similar estimation are the lactometer and

creamometer devised by Dr. Holt. With these we may examine a smaller quantity of mother's milk ( $1/2$  ounce). But, in the main, we prefer the above-mentioned method (page 102), for it obliges us to empty the breast, and we may so obtain a greater amount of information, and possibly more accurate knowledge. It is also necessary that the creamometer should stand for twenty-four hours, while the centrifugal method permits us to read the percentage of fat immediately. If a creamometer is used, we must read 3 per cent. fat for every 5 per cent. of cream.

The reaction of human milk is rarely studied, though most authorities state that it is faintly alkaline. Holt contends that "even when freshly drawn" it is "amphoteric to litmus and acid to more delicate tests (phenolphthalein)."

Microscopically, we may find fat globules (see above), colostrum corpuscles, pus and blood (Holt). The last two must always be considered abnormal. In cases of mammary abscess or sepsis in the mother, it is wise to submit samples of milk to a skilled bacteriologist. If found after a fortnight's time, colostrum corpuscles are viewed as abnormal.

**Advantages Possessed by Human Milk when Compared to any other Infant Food.**—These are: 1. Physical. 2. Chemical. 3. Biologic. 4. Clinical. 5. Sociologic.

1. The well-known physical advantage relates to the flocculent curd produced when the caseinogen of human milk is acted upon by rennin in the presence of hydrochloric acid. The casein of cow's milk forms a solid curd (see the table from Chapin given below).

Types of milk	Animal	Curds	Water %	Fat %	Sugar %	Pro-teids %	Salt %	Month when puberty is reached
Carnivorous (stomach 60-80 per cent. of digestive tract).	Dog....	.....	75.44	9.57	3.19	9.91	0.73	6-8
Ruminant (herbivorous stomach 10 per cent. of digestive tract).	Sheep..	Solid.....	83.50	6.14	3.96	5.74	.66	6-8
	Goat....	Solid.....	86.91	4.09	4.45	3.69	.86	6-8
	Cow....	Solid.....	87.17	3.69	4.88	3.55	.71	8-12
Non-ruminant.....	Mare...	Gelatinous.	90.06	1.09	6.65	1.89	.31	18
	Ass.....	Gelatinous.	90.00	1.30	6.30	2.10	.30	18
Herbivorous (intestine 90 per cent. of intestinal tract).	.....	Flocculent.	88.20	3.30	6.60	1.50	.20	14 years.

2. It possesses manifest chemical advantages, as we trust has been sufficiently shown. Its chemical constituents differ so much from other milks that it may be said to possess a specific composition. Chapin's table reveals this very graphically.

3. Biologically human milk is infinitely superior. The breast-fed baby increases in weight more steadily and consistently than does his bottle-nurtured contemporary. Again, as Chapin insists, the specific milk best prepares the young animals gastro-intestinal tract for its future work in life.

The young antelope and the young big-horn in their respective play-lives have both been observed to indulge in repeated leaps. But the antelope leaps forward, while the big-horn leaps straight into the air (Charles Darwin). Both animals while thus rehearsing useful activities in the histories of their species, are inadvertently preparing themselves for the contingencies of their future lives. So the milk of the bitch is preparing the puppy's gastro-intestinal tract for the food of a carnivorous animal; the milk of the cow is suited for the needs of a ruminant animal; that of the mare for the non-ruminant herbivorous animal; that of woman for the omnivorous animal. (See Chapin's table, just cited.) More recent work has shown that the blood of the naturally nourished baby has a higher bacteriolytic power than the blood of the baby fed solely upon the bottle. We have called attention previously to the fact that alexins of the mother's blood pass over in the milk.

4. The clinical advantages of nature's way have long been apparent to observant physicians. Some of these are:

*a.* There is less gastro-enteric disturbance of a functional nature (indigestion) among breast-fed babies.

*b.* There is less danger of gastro-enteric infections. The summer diarrheas that so decimate our infant populations are almost confined to artificially fed babies.

*c.* As mentioned previously (biologic advantages), babies gain more consistently and more rapidly on the breast.

*d.* Rickets, atrophy, and other evidences of innutrition are not so common in maternally fed babies; unless lactation be continued too long. Scurvy is very rare in breast-fed babies.

*e.* Breast feeding on the one hand, or necessary dependance upon the bottle on the other, may actually determine whether the delicate baby lives or dies. This is particularly true of babies with a luetic inheritance.

5. Sociologic advantages are possibly not germane to a med-

ical discussion, but we feel that they should be duly recognized by every broad-minded medical student and physician. It is generally recognized that "the family is the unit of the state," just as the individual is the unit in the family life. What an important service has been rendered the family and state when a citizen in embryo has been supplied the best nourishment at what is unquestionably the most important nutritional epoch of his career!

From the maternal side, viewed in its most practical, sordid light, breast-feeding is less trouble than properly conducted hand feeding. Lactation, like pregnancy, also means, or should mean, rest from the sexual life.

*Ethically*, lactation may necessitate unselfish devotion to the baby and to the hygienic life; but if this reason be adjudged a sentimental one, it is still sentiment that rings true. With modern knowledge, mothers may make of maternity a noble profession.

#### RULES TO GUIDE IN BREAST-FEEDING

1. **Hours for Nursing.**—After the completion of the labor, the mother should be allowed a rest of from four to six hours. The average baby tends to sleep more deeply during the first twenty-four hours of extra-uterine existence than he will at any subsequent period of infancy. He should be aroused at four-hour intervals and placed at the breast, clean water being given him before every nursing. On the second day of life he should be fed every three hours from 7 A.M. to 10 P.M. and once during the night.

The modern tendency is to keep the sturdy baby upon this three-hour interval. But we are purposing to feed individual babies according to their individual needs, and some babies will require feeding every two hours.

If the latter is the case, we change to a two and one-half hour interval when the baby is somewhere between two and six weeks old. The time to change will depend upon the baby. At three or four months, we change to the three-hour interval, giving but one feeding at night. (See table under artificial feeding.) Some authorities change later to a three and one-half and even four-hour interval. We are inclined to favor the three-hour interval, gradually doing away with the night feeding and the 10 o'clock nursing. We are accustomed to speak of our three sevens; *i.e.*, a baby of seven months should sleep from 7 P.M. to 7 A.M. This long period of infantile digestive rest means also a splendid period of rest for the mother. But, there are babies and

babies, and it is ever to be borne in mind that we are feeding individuals.

2. **The Care of the Nipples.**—We shall have nothing to say concerning methods preparatory to parturition; but we do wish to emphasize, from the infantile standpoint, the importance of scrupulous cleanliness. The nipples should be washed before and after nursings and protected by sterile cotton or gauze between the nursing periods. The inverted nipple may require the use of the nipple shield, as may also the fissured nipple. For weak and premature babies, it may be necessary to employ the shield, or possibly the use of a Breck feeder after the breast has been emptied. The method of securing breast milk in a cleanly manner has been described.

3. **Clock-like regularity of feeding** should be followed. "The clock, and not the baby's cry," should indicate the feeding time. The necessity for such system is well recognized in our own lives, and is equally recognized in the raising of live stock. *A priori*, it would seem still more important in dealing with immature organisms. As a matter of common observation, there is probably no cause of infantile digestive disturbance so prevalent and so potent as irregular feeding.

The baby should be roused at regular intervals during the day, and allowed to sleep for longer and longer periods during the night. If he is not roused during the day, he tends to be wakeful at night. Humorously, though truthfully, it has been stated that the human baby shows feral tendencies and tends to prowl and growl at night.

4. **How long a time should be allowed for a single feeding?** In general we may state from ten to twenty minutes. But, again, we must recall that we are prescribing for individual babies with individual milk supplies. The milk supply may be more than sufficient or it may be barely enough; the lacteal ducts may be of large or small caliber; the baby may be strong or weak. Thus one baby may secure in five minutes what it will take another four or five times as long to secure. Probably the best criterion is furnished by weighing the baby before and after nursing.

5. **The "let alone system"** or plant life for the baby: Infancy is the age for true kindergartening. We should endeavor to supply the proper conditions for growth and development, as in the case of the plant; and for the rest, we trust to good Dame Nature. The baby under such treatment tends to become a healthy, happy little automaton.

We find it very necessary to be very explicit with parents, as Dr. Holt is explicit with his nurses. A statement like the following con-

veys far more than any generalizations: "The baby should be picked up for the following reasons and these alone: to be nursed and given water; to be bathed; to have his napkins changed; to be dressed and undressed; to be given his airings and exercises. He should never be picked up simply because he cries."

6. **Hours of Maternal Rest.**—In general, nursing mothers need more rest than other women. They should have at least eight hours of night sleep, and one or two periods of complete relaxation (better, sleep) during the day. A rest of an hour in the forenoon and of another in the afternoon amply repays the mother for the time she may consider lost.

7. **The Maternal Diet.**—This should be ample and nutritious. It should include milk, eggs, meat (once a day), green vegetables, fresh and cooked fruits, cereals and other starchy foods in moderation, and simple desserts. In general, stimulants, indigestibles, condiments, highly seasoned foods and excessively sweet substances should be avoided. It may be put down as an important generalization that foods that do not disturb the mother's digestion will not disturb the baby (Griffith). We shall have more to say on this subject, however, in discussing the reasons for failure in maternal feeding. In many cases, it is well to apply the calorimetric method to the mother's diet.

8. **Exercise and Fresh Air.**—Most nursing mothers should indulge in less exercise than has been their former wont. This applies particularly to the athletic girl. Energy expended in muscular exercises may be energy diverted from glandular activity. Every intelligent stock breeder appreciates this principle; but how little it is applied to or by mothers. Walking, in moderation and in the open air, is one of the best exercises. Under certain conditions, shortly to be mentioned, it may be necessary to take more exercise, even to the fatigue point. On the other hand, it may be necessary for the nervous mother to take more rest (see below). How often one hears the remark "I lost my milk as soon as I got out of bed."

9. **The Maternal Bath.**—Whatever may be said of exercise, and its amount should be regulated for the individual, there is no doubt concerning the need of the daily bath for every mother. It secures not only a cleansing effect, it stimulates not only the skin and other emunctories, but it also secures a reflex stimulation of all metabolic processes. For mothers at all vigorous, we prefer the warm cleansing bath, followed by the cold sponge or shower, and the brisk rub with a Turkish towel.

**10. Regulation of the Mother's Bowels.**—We shall not here enter into ways and means, but shall simply say that the attending physician should see to it that the nursing mother has a free daily evacuation of the bowels. If she does not, he should see that proper measures are instituted to secure such regularity.

**11. Drugs that may be Excreted in Milk.**—Much more should be known about this subject than is known. One of us saw a marked bromide rash on a two days' old baby. The mother had been given ethyl bromide as an anesthetic during her labor. The drugs that are positively known to pass from mother to child through the milk are: the saline cathartics (and some vegetable aperients); belladonna; the iodides and bromides; mercury; arsenic; antimony; opium and iodoform (even when used externally by the mother). Alcohol, when freely indulged in by the mother or wet nurse, may disturb the suckling very markedly, though Holt states that its excretion in breast milk has not been proven.

It is claimed by most authorities that the drugs mentioned are more liable to be eliminated in breast milk when the milk is scanty and poor.

The excretion of mercury through the mammary channel is not to be depended upon in the treatment of inherited lues, as it is somewhat uncertain. Opium and its alkaloids have caused serious symptoms and even death in the young baby; but it is maintained that they pass over in small amounts ordinarily, and that the mother must either take large doses or produce a poor milk to endanger her child.

In conclusion, we must reiterate that much more should be done to place this important question upon a scientific basis. Meanwhile, we quite agree with Still that the fewer powerful drugs given a nursing mother, the better off will her child be. Alcohol, we absolutely avoid, unless it be given in the form that we shall shortly mention.

**12. Weaning.**—In the past this word was almost a synonym for trouble in the nursery world. There is little doubt that the old-time fear of the second summer—a well grounded fear in fact—simply meant that babies in their first summer were usually breast-fed, while in their second year they were exposed to the dangers of hand feeding. Nowadays, the baby who is bottle-fed from an early period is exposed to greater dangers in the first summer than he is in the second.

Judicious treatment from the first may rob weaning of practically all its terrors. How? In the first place, no matter how good the breast milk proves, a single bottle should be given from an early period in infancy (Holt). This permits of experimentation in the food line, and sometimes permits us to correct certain serious faults in the mother's

milk. The baby who has learned to take one bottle will not object to taking several. Not a small benefit accrues to the mother from this bottle, for she is allowed more leisure and more liberty. In the second place, we must carefully determine the time for weaning. Few American mothers successfully nurse their babies for more than eight or nine months. Still others can nurse successfully for only about three months' time. As has been said, however, a half loaf is better than none, and if such evidences of depreciation in quality or amount should appear during an acute illness of the baby, or during the summer season, we still strive to maintain some of the maternal supply. Thirdly, if as advocated, a bottle has been given from the first, weaning may usually be effected gradually. We usually supplant one more breast feeding a week by one more bottle, until the breast is gradually and safely deserted. In the old days, this gradual substitution was advocated; but it was often impossible to follow out. The mother had to absent herself from the baby in order that the bottle would be taken at all. In our own experience, one baby refused to take a bottle for three weeks, though she subsequently did beautifully on her milk formulæ. We see no reason to quote special formulæ in this connection. The principles of such prescribing will be dealt with under bottle feeding.

### THE REASONS FOR FAILURE IN MATERNAL FEEDING

1. **The Colostrum Period.**—This is a dangerous shoal upon which the ship of well-intentioned maternity often founders. Colostrum, high in the proteids, may cause marked digestive disturbances (intestinal colic) or even metabolic disturbances (accompanied by high fever, etc.). The baby's crying upsets the nervous mother, and her nervousness in turn increases the protein content of her milk. And so we have a vicious cycle initiated. If the mother's determination weakens, and she nurses the baby irregularly, matters may easily proceed from bad to worse. The best plan is to keep the baby in a distant part of the house; to feed him less frequently, not more, and to give him water or other dilutents (see below) before his nursings.

2. **Deficient Supply of Breast Milk.**—This may constitute a real difficulty; but, on the other hand, it may represent only late arrival of the milk. A number of times we have seen milk come in plentifully on the fifth day or later. In one remarkable instance, in our own experience, the wife of a distinguished anatomist, a primipara in her thirties, developed a plentiful supply of breast milk on the twentieth day of lactation. We were able to desert our hand feeding in this case,

and did not have to resume it again until her baby was nearly four months old.

As we have already indicated, the most practical way of determining how much a baby gets at a nursing is the simple method of weighing him before and after he feeds.

In general we feel that the breast supply should never be deserted in less than two weeks simply because it seems deficient in amount.

**3. Lack of Development or Anomalies of the Mammary Gland.**—This includes atresia of the ducts; undeveloped or distorted nipples, etc. Of course atresia of the ducts renders breast feeding impossible. One of us was called by Dr. John C. Hirst in such a case. The mother, a physician's wife, had an enormously distended breast, and it was deemed imperative that the baby be fed in another way.

On the other hand much may be done for inverted nipples by proper treatment, and by judicious use of the nipple shield. As is well known, fissured nipples may usually be healed without desertion of breast feeding.

**4. Mammary Abscess.**—This sometimes raises some nice problems that concern both mother and child. Feeding may sometimes be carried on with the unaffected breast, and this may either be partial feeding in association with substitute feeding or it may itself yield a sufficient supply. The affected breast should not be nursed until it is positively determined that its milk contains no pus organisms.

**5. Puerperal Infections.**—These again bring up serious questions that must be judicially weighed, from the maternal and infantile standpoints, in order that they may be wisely answered. From the infantile standpoint, chemical analysis and bacteriological examination of the milk furnish the best criteria. As a rule, in the interests of both mother and child the patient with puerperal sepsis should not nurse her child.

**6. Acute Febrile Diseases of the Mother.**—Typhoid, pneumonia and some other diseases may affect the quality and quantity of breast milk. Again, the mother may be so ill that lactation represents a dangerous drain on her strength. If the disease is of short duration, temporary hand feeding may tide the baby over for a few days, when the breast feeding may be successfully resumed. In the presence of typhoid fever, it is usually best to cease our efforts at nursing the baby. In the presence of diphtheria in the mother, the baby should be given antitoxin.

**7. Puerperal Eclampsia.**—This is probably a very real danger. Goodall has made out a very strong case in describing three babies,

who shortly after nursing from eclamptic mothers, developed similar symptoms and died. He advises that the baby should be fed on some other food for several days after the mother's symptoms have ceased. Her breasts should also be emptied after the eclamptic symptoms have subsided. Esch and others contend, however, that such babies are affected prenatally.

**8. Maternal Poverty with Insufficient or Improper Food.—**

The nursing mother should have the best of food and that in abundance. Starvation may cause the cessation of lactation. Nevertheless, it is at times wonderful to see how nature conserves her supply of milk for the baby, even though the mother's nutrition suffers sadly in the process.

**9. Constitutional Diseases.—**Under this heading we shall speak particularly of tuberculosis and syphilis. The mother with active tuberculosis should never nurse her infant. To do so usually means disaster for herself and for her child. Tubercle bacilli may not appear in milk unless the breast is affected by the disease; but the milk is usually poor in quality. On the other hand, the syphilitic mother, or the apparently healthy mother who bears a syphilitic child, should always nurse her baby if it is at all possible for her to do so. The baby is an example of Profeta's law just as the mother is of Colle's law.

**10. Excessive Nervousness.—**Probably in this cause and in the one to be next mentioned, we have the explanation of most failures in lactation. Nervousness may manifest itself in numerous ways, just as its causes may be numerous. There is some vice of nervous organization resulting from heredity or past environment or both, and there are also exciting causes, among which the baby's crying may not be least. The influence of the emotional storm—passion, fright, etc.—upon the milk may be most profound. Rotch contends that the proteids are increased, and the fats diminished. But no matter what the exact chemical changes, the milk disturbs the baby, possibly seriously. The nervous mother is liable to make a poor nurse.

**11. Too Many Responsibilities, Physical and Mental.** (Usually of the domestic and social nature.)—In discussing the question of activity and rest, we have already called attention to an important law: Energy expended in muscular or mental activity must of necessity mean less available energy for grandular functions. The farmer's boy who runs a milch cow from the field usually receives a well-deserved punishment—one that should teach him a valuable lesson. But how often we find nursing mothers driven and harried by countless

duties and unnecessary irritations. We cannot and do not expect the modern mother to lead a colorless bovine existence, and yet for the time being successful child rearing is her most important duty in life, and the most important problem of the baby's existence is its nutrition. The whole intellectual atmosphere of the mother's life is transformed when she once learns to view maternity as a profession.

We fail to find many women who do not wish to nurse their progeny, and contrary to frequently expressed views, we do not find these as often in the higher as in the lower walks of life. Those mothers whose thoughts fly bottleward are generally inexperienced young matrons, as immature in mind as in years. We find far more mothers who would willingly nurse their babies if they only could.

12. **Return of Menstruation.**—This is one of the “bug-a-boos” of the nursing world. The great majority of mothers secrete normal milk even though menstruating. If there is disturbance of the mammary functions and resulting disturbances of infantile digestion, such phenomena are usually only slight and temporary. Rotch and others have described the changes that may take place in the milk of the menstruating mother, and there has been much discussion of the whole subject pro and con. But we shall simply say that the mother should not be led to anticipate detrimental changes; and if perchance her baby is disturbed, the problems of substitute feeding (temporary or permanent), partial breast feeding, etc., are problems that must be worked out in the individual case.

The breast milks of 180 women were examined by Jacobius after six months of lactation; 51 per cent. of the mothers were not menstruating. During menstruation he could not find any chemical changes that would seem to make weaning necessary. Twelve babies lost weight temporarily, were restless, and had dyspeptic stools.

13. **Pregnancy** should lead to prompt cessation of breast feeding, unless the baby at the breast is suffering from some transient disorder which might make immediate weaning a hazardous procedure.

14. **Heredity.**—Aaron Jacobi and others have contended that this is an important factor in determining the inability or ability to nurse. The mother who cannot nurse her baby is often the daughter of a mother who has failed to nurse hers. Some statistics have seemed to support this contention. But, viewing the problem in a broad biologic light, one would be forced to view inability to nurse as an artificially acquired characteristic. One would consequently expect, *a priori*, that such a characteristic would tend to disappear under normal conditions of life. We feel very strongly that this problem

will be answered as soon as its fundamental importance to the race is recognized—when human thought will be directed toward its solution with the same seriousness of purpose that is evidenced in the solution of many biologic problems.

15. **The insufficient supply fails to satisfy the baby;** there is infantile indigestion, or, despite a large supply of milk, the baby does not gain in weight. These are real difficulties, of every day occurrence, and must be met. If the medical attendant will only recognize the vast superiority of mother's milk when compared to other infant foods, if he will only appreciate the importance of successful lactation to the family and to the race, he will never decide nor act too hastily in the presence of such conditions.

As previously stated, a deficient supply in the early days of lactation may spell only a late arrival of the milk. In addition to the mother's bountiful and nutritious food, fluids should be given her in plentiful amount. Milk is probably the best; but cocoa and broths may also be allowed to vary the monotony of her diet. In general, it is best to give these fluids, particularly milk, between the regular meals (on awakening, in the middle of the morning, in the middle of the afternoon, and at bedtime). Corn-meal gruel (Southworth) seems to be another helpful article of diet. The value of malt preparations has been discussed for a number of years, but in a few cases Holt has shown that malt increases both the amount of milk and the fat percentages therein. Massage of the breasts may also prove of service.

If, after several weeks or months of successful nursing, the mammary secretion becomes insufficient for the baby's needs, it is always to be borne in mind that "a half loaf is better than none." To the observant mother, the baby usually proclaims this difficulty eloquently. He probably nurses voraciously for a short period of time. Then, dropping the nipple from the mouth, he evidences his disappointment in loud crying. If he is weighed before and after the nursing, it is found that he secures a totally inadequate amount. The difficulty may be met in several ways: He may be permitted to nurse from one breast at a time and may then be fed a proper milk formula in sufficient amount to atone for the maternal deficit. Again, he may be fed from both breasts and be given a milk formula at his next feeding-time.

If the baby suffers with gastric or intestinal indigestion or both, attempts may be made to secure relief by treating both mother and baby. Despite much recent work, we believe that in most of these cases the milk proteids are in excess (proteid indigestion or intestinal

"colic"), and that in a certain smaller proportion of cases, excessive fats are at fault (butyric acid fermentation, with gastric or upper intestinal indigestion—Westcott). In still other cases, both of these solids may run too high. Analyses are helpful, as already indicated. Unless the mother is exceedingly neurotic, she should be made to exercise more than has been her habit, and the amount of proteid in her diet should be lessened. If she is neurotic, more rest should be advised; she should be fed freely and given tonics (compound sumbul pill, etc.). The baby should be fed less frequently, and fluid should be given him before he nurses in the hope of diluting the milk (Rotch). Personally we run the gamut of three diluents: 1. Boiled water; 2. half-strength barley water; 3. equal parts of barley water and lime water. Usually we employ these diluents in the order mentioned, and ordinarily we administer two drams of one or the other before every feeding. With the third diluent, one suggested by the late Frederick Packard, we have often seen the number of stools reduced; have seen them change from a green to yellow, and have found the babies relieved of pain. But occasionally these methods fail. It is then our custom to give a digestant before the feeding: One that has yielded the best results is:

R. Pancreatin..... gr. i  
 Soda bicarbonate..... grs. iii  
 Dispens. xxx such powders.  
 Sig.—Give one powder before every feeding.

Some authorities have even drawn the mother's milk and peptonized it. This method we have not employed. One mother spoke of the powder just mentioned as the best sedative ever given her baby.

Starr calls attention to the fact that the "colicky" baby often thrives. The constant crying seems to hurt the parents more than the baby. But no matter how stout the baby grows, his nervous nutrition must inevitably suffer. We quite agree with the authority who said that the "three" or "six months colic" should be considered a three or six months disgrace.

If, despite a sufficient supply of milk and an undisturbed infantile digestion, the baby does not gain in weight, recourse should again be had to an analysis of the milk. It may show a product hopelessly deficient in solids. Once more we may advise rest of body and mind for the mother; once more in our efforts to aid her we may resort to forced feeding; but too often such efforts are destined to fail. The baby should also be fed more often, as this tends to increase the solid content of the milk (organic secretion).

**Wet Nursing.**—In considering the advantages of human milk, it has been said that there are cases in which no other food will save the infant's life.

The ethical medical man, therefore, cannot escape the conclusion that wet nursing is sometimes a necessity. Some of the Continental countries are far ahead of England and America in their recognition of the importance of this problem. The registering of wet nurses in this country would represent an enormous advance in our sociologic system.

One of us shall never forget a baby seen by Dr. Griffith and himself in the practice of Dr. Guthrie of Philadelphia. The baby was a twin and his brother was dying at the time of the consultation. He was miserably atrophic, and had had an acute gastro-enteric infection. Even the determination to try breast milk seemed a very dubious experiment. The consultation was held late at night, and we were unable to secure a good wet nurse until late the following afternoon. In the meantime some of the mother's friends—young mothers nursing their own babies—arrived in relays. In the morning 1 dram of breast milk, so secured, was fed the baby by a medicine dropper. He was then exhausted. By evening he was able to take 7 drams, fed to him in the same way. On the morning following, we watched him suckle at the wet nurse's breast for twenty minutes. He is now a very sturdy boy of six years.

When the question of wet nursing is raised, the chief point to be considered is that of common justice to all concerned: 1. The baby to be fed; 2. the wet nurse; 3. the wet nurse's child. All have rights, and these should be carefully weighed. In a nearby city, the rights of the wet nurse were woefully disregarded. She was called upon by the municipal "Poor-board" to help nurse an atrophic baby. She developed a chancre of the nipple, and before the nature of her malady was known to her, infected every member of her immediate family.

**The Difficulties of Wet Nursing.**—These are many and at times they are or seem to be insuperable. The principal ones are: 1. The difficulty in securing a proper wet nurse; 2. the question of expense; 3. the difficulties that arise in the household; 4. the dangers of disease from the nurse; 5. the possible habits of the nurse; 6. the fact that the baby may not take to the breast; 7. the wet nurse's milk may fail to agree with the baby. Briefly we shall proceed to consider these obstacles seriatum:

1. *The Difficulties in Securing a Proper Nurse.*—Anyone who in his anxiety has telephoned to hospital after hospital only to receive negative

or indifferent replies need not be told how difficult it may be to get a wet nurse. The difficulty would be minimized if every maternity hospital and every hospital supporting a maternity ward would ask of every applicant for admission: "Are you willing to act as a wet nurse?" Subsequently, the hospital physicians would be in a position to study both the mother and her baby; and if the study showed the mother fit for the task, she should be registered as a wet nurse. If another baby in the institution needed wet nursing, and some of her yield could be utilized without injustice to the woman's own child, she should never be merely asked to furnish some milk. It should be demanded of her. She would thus repay in some part her debt to society.

2. *Expense.*—In Philadelphia a wet nurse receives from ten to fifteen dollars a week. This sum represents more than many families in the humbler walks of life can afford. Recourse may here be had to partial wet nursing, or to the use of breast milk secured in sterile bottles.

3. *The Household Difficulties.*—The wet nurse occupies an anomalous position in the household. With all due consideration for her value and her sacrifice, it must be said that she often becomes the traditional beggar on horse-back. The knowledge of her power may make her a czar, and her ignorance may make her a dangerous and vindictive dictator. Thus, a wet nurse in the family of a prominent druggist rendered for a time splendid service to twins. Then realizing her power and their absolute dependence, she refused to nurse them at night. She said she must have unbroken rest. The stated need did not prevent her from awakening nightly to indulge in alcoholic libations, however, and she was dismissed.

4. *The Danger of Disease.*—Still records three cases that graphically illustrate this danger. One wet nurse, successful for a while, died of miliary tuberculosis. Another had pediculosis. A third, apparently healthy, had a syphilitic infant.

5. *The Habits of the Wet Nurse.*—Among detrimental habits, alcoholism probably crops out most frequently. Sexual immorality may also represent a serious menace. One of the best wet nurses we ever employed, so far as her mammary yield was concerned, was denominated by the head of the family employing her as "not immoral but absolutely unmoral." In addition to these grave menaces, women employed as wet nurses may visit and feed their babies surreptitiously, may indulge in forbidden articles of diet, and may sedulously conceal the existence of so important a condition of body as constipation.

6. *The Baby may not Take the Breast.*—At times, this seems a very real difficulty, but usually patience and ingenuity bring eventual success.

7. If the nurse's milk does not agree with the baby, an analysis may reveal the cause of the trouble. The rules laid down in the study of mother's milk may serve us, and in the presence of minor disturbances of the infant, we may secure results by treating both wet nurse and baby (see above). If we fail along these lines, however, our only alternatives are to secure another nurse or to employ bottle feeding.

**Points to be Observed in the Selection of a Wet Nurse.**—

Taken all in all the best index is the health of the wet nurse's baby; but there is only one safe rule to follow: *Examine or have examined both the wet nurse and the baby, and see that both are examined from the crowns of their heads to the soles of their feet.* Pediculosis; cutaneous diseases or scars; mucous patches in the mouth; contagious diseases of the eye; affections of the throat; pulmonary disease; mammary abscess; cardiac disease; fissures of the nipple or distortions of the nipple; adenopathies, vaginal or rectal condylomata or vaginal discharges are the principal conditions to note in the mother. In the baby we note particularly the state of nutrition, and presence or absence of congenital luetic phenomena. We would strongly advocate the employment of the Wassermann reaction in every applicant for the position of wet nurse.

The shape and size of the wet nurse's breast may prove deceptive, and wet nurses may be malingerers. Those wishing to escape such service in German Hospitals have often emptied their breasts before the examination; while those desiring employment have permitted their breasts to become greatly distended. An examination of the breast milk should always be made.

After carefully following these rules, however, we may find that an apparently perfectly normal milk, agreeing perfectly with the wet nurse's baby, will still fail to agree with the baby to be nursed.

**Partial Wet Nursing and the Use of Bottled Mother's Milk.**—

Some hospitals systematically employ the former method, utilizing either their nursing inmates or poor mothers in the neighborhood. The latter are paid so much per ounce for their yields. We know of several cases, in which physicians have induced nursing mothers among their poor patients to suckle other babies two or three times a day.

Breast milk may be secured in sterile milk bottles by the method already advocated. Poor mothers with plenty of milk will usually be glad to furnish such milk for a suitable consideration.

In conclusion it must be said that wet nursing, with all its difficulties, represents a very important type of infant feeding—one too often forgotten or neglected; and that in its pursuance, one should ever have before him the eternal question of justice to all concerned.

## BOTTLE OR HAND FEEDING: ARTIFICIAL FEEDING

If we except the congenitally deformed, the congenitally diseased the babies injured in birth and the babies infected at birth, or shortly after it, very few breast-fed infants die. *A priori*, therefore, it would seem best to attempt to follow nature's way when for one reason or another a baby cannot receive its natural food. In these efforts also it would seem logical to be guided by two principles: 1. Mother's milk is practically sterile; its substitute should be clean. 2. Mother's milk possesses a more or less definite percentage composition of its organic constituents; its substitute should furnish percentages of proteids, fats and carbohydrates suited to the baby's digestive ability and nutritional needs. Anent of this second principle, it will be admitted by every student that he is not attempting to supply the same proteins and the same fats; but if he furnishes *milk* proteins and *milk* fats in proper proportions, he is prescribing better food than can be furnished in any artificial product of the manufacturing chemist.

In this book we have devoted considerable space to breast feeding. We shall make no effort to present an exhaustive treatise upon hand feeding. Indeed, years of endeavor have convinced us that we gain most by presenting the latter subject as simply as possible to medical students and general practitioners.

### The Securance of a Clean Food.

**A. Sterilization.**—This word, which means rendering sterile or free from germs, is rather "loosely used" in its relation to milk. Usually milk is heated to  $212^{\circ}$ , and kept there for an hour or an hour and a half. It is then cooled and placed on ice. Sterilization is preferably done in small bottles, stoppered with sterile non-absorbent cotton. In such milk, spores are not all destroyed, and the product cannot be kept indefinitely. If milk is taken on a long journey, it should be so sterilized for several successive days (fractional or intermittent sterilization).

In this country the "Arnold sterilizer" is the apparatus most commonly employed for this purpose.

Sterilization of milk is still largely used on the Continent of Europe, but it has certain disadvantages: Lactalbumin begins to coagulate at 160° F.; casein is rendered less digestible; certain alterations take place in the fats; lactose is changed into caramel; organic phosphorus is transformed into inorganic phosphates; citric acid is partially precipitated as calcium citrate; and some lime salts are converted into insoluble compounds (Holt). Whatever the exact chemical changes, however, the nutritive qualities of the milk as well as its digestibility may be seriously affected. It produces scurvy in a number of instances (117 cases in 379—Report of "The American Pediatric Society, 1898").

Sterilization, however, must be accorded certain spheres of usefulness: In the preparation of milk for long journeys, and for the consumption of the babies of the poor (particularly in warm weather), it may furnish the safest food.

**B. Pasteurization.**—This process really implies two procedures or steps: The rapid heating of milk and its maintenance at a given temperature for a certain time; and subsequent rapid cooling of the milk.

The original temperature to which the milk was raised was 167° F. (75° C.). It was maintained at this point for from twenty to thirty minutes, and was then quickly cooled. At a later period 155° F. (68° C.) was accepted as the proper pasteurizing point (Freeman, Russell, etc.). At the present time 140° F. has been shown to be sufficient to accomplish the purpose of pasteurization. The milk is maintained at this point thirty to forty minutes (Russell, Freeman, Rosenau etc.), and is then quickly cooled. At the temperature last mentioned, the ferments of cow's milk are not destroyed, and so far as we know, no important chemical changes take place in the milk. On the other hand, most of the pathogenic bacteria are destroyed. It is fully recognized that spores are not destroyed, that pasteurized milk should be kept on ice, that it should be handled more carefully than non-pasteurized milk, and that it should not be used after twenty-four hours. Preferably, milk should be pasteurized in the nursing bottles, and not in jars. The organisms that produce souring of milk (*bacillus acidi, lactici*, etc.) are readily destroyed by this process. We are thus robbed of an important index of the age of the milk. But in milk which is still sweet, pathogenic bacteria may have multiplied to an astonishing degree. So great an authority as Conn, of Wesleyan University, thinks that in pasteurization we have our best present answer to the milk problem.

The Freeman apparatus or that sold by the Walker-Gordon Laboratory Company are ingenious and convenient, though somewhat expensive devices for home pasteurization.

We have no wish to belittle the great milk charities which at no cost or at a nominal cost supply pasteurized milk, or modified pasteurized milk to the poor; but the public should know more than it does concerning commercial pasteurization.

There are some plants that are really equipped to pasteurize milk in the jars in which it is dispensed; but most of the so-called pasteurized milk sold by milk dealers is not pasteurized at all. One large dealer said to one of us: "We realize that the process is not pasteurization. It is the 'shocking process.'" We have been grateful to him for that term. Its subsequent sale may also be denominated a "shocking process," for the layman buys what he supposes is a pasteurized milk. Is it not called so? More than that, it must be more carefully cared for than ordinary market milk. As we have already mentioned, the souring organisms have been destroyed and the purchaser is robbed of this important indication (souring) of the age of the milk (Cope and Evans). Nevertheless, it may become infested with pathogenic germs. Live tubercle bacilli have also been found in commercially pasteurized (?) milk. "Commercial pasteurization is often a commercial fraud."

**C. The Certification of Milk. Certified Milk.**—When a milk measures up to certain standards of production, guaranteed by veterinary inspection; when it comes up to certain chemical requirements, as proved by chemical analysis; when it meets the requirements of bacterial cleanliness, as verified by the bacteriologist, certificates are granted to the dealer or producer. In this country a milk so produced and safe-guarded is styled a "certified milk." The father of certified milk is Dr. H. L. Coit, of Newark, N. J., and we may well be proud of this "American idea." The certificates are furnished by some disinterested body, such as the "Medical Society of the County of New York," or the "Pediatric Society" in Philadelphia. These certificates are so placed on the sealed cap over the mouth of the milk jar that the consumer may readily see them. Certified milk is now obtainable in many large cities and summer resorts of the United States.

The various rules laid down by milk commissions that supervise the production and sale of "certified milk" we shall not go into at great length. Chapin puts it well when he says: "Eternal vigilance is the price of low bacterial count."

The veterinarian must see that the cows are tuberculin tested, and are free from other diseases such as mastitis. He must see that they are kept clean by daily grooming and that their udders are carefully washed and dried before milking. He directs the diet of the cows. The cow barn must be light and well ventilated. Its flooring must be well made (cement or wood); its drainage must be good, and the floors, walls and ceiling must be kept scrupulously clean. Dust must also be excluded by attention to fodder, bedding and methods of cleaning the barn (moist mops, etc.). Sick animals and calves are excluded from the cow barn. The milkers must wear clean white suits, and their hands must be scrubbed carefully before engaging in milking. They must be free from contagious disease, and not permitted to come in contact with patients suffering with diphtheria, scarlet fever, etc. Milk vessels and utensils must all be sterilized in a steam sterilizer, etc. The milk is immediately cooled and bottled at the farm. The veterinarian makes his examination at least once a month.

The chemist and bacteriologist also make monthly examinations, more often if deemed necessary. Most of the commissions require that the butter fat shall attain 4 per cent. and some of them also certify 5 per cent. milks. Samples of milk for examination may be taken from the milk depot or the milk wagon, and the dealer does not know when they are to be taken. The bacterial standards are different in different localities, some commissions permitting 100,000 bacilli to the cubic centimeter; some 30,000 (New York), and some only 10,000 (Philadelphia).

There can be no question of the fact that up to the present time "certified milk" is the best form of bovine milk that has been produced for infant consumption. The sociologic difficulty that it presents is its cost of production and its consequent cost to the consumer. Nevertheless, it is wonderful to see what sacrifices humble people will make when they once understand the difference between this and ordinary milk. Again, the striving for the ideal that its production necessitates has already had an enormous educational influence upon farmers and milk dealers.

Chapin contends that local conditions of milk production should be studied before restrictions and regulations are laid down, and that farmers and dealers should be made to realize that they are being helped and not harassed in the effort to produce clean milk.

*The Securance of Clean Milk for a Baby Summering on a Farm.*—It is surprising how much may be attained from simple material at hand, and that by little effort. For several years we have used the following

method with signal success: The mother is instructed to tie four layers of cheese-cloth around the neck of a quart preserving jar. The amount of cheese-cloth used is ample enough to permit it to intrude into the jar in the shape of an inverted cone. Daily, before milking-time, she is to boil the jar, cheese-cloth and screw-cap of the jar. The cap is then to be placed over the top of the jar. She is then to carry her sterilized utensils to the barn at milking-time. Arrived here, she is to pour some of the mixed milk (from the herd, or from several cattle) through her sterile cheese-cloth into her sterile jar. Then she is to cut the string that holds the cheese-cloth, throw the latter away, and screw on the sterilized screw-cap. The jar is then ready to be placed on ice. If the herd is not tuberculin tested, the milk is to be pasteurized in the jar or in the individual nursing bottles. The mother takes all the trouble, and consequently she is not opposed by the farmer. If she meets with such opposition it may easily be overcome by a small additional emolument.

**II. The Modification of Milk to Meet the Baby's Digestive Ability and Nutritive Needs.**—In the consideration of this subject it is necessary to give passing notice to the use of milk from animals other than the cow. Mare's milk is difficult to obtain; but is said to agree splendidly with some babies. This is probably explained by its low fat and proteid contents, and the gelatinous curd resulting from the action of rennin upon its caseinogen (see biologic table from Chapin, page 104). Asses' milk is used in Paris among the wealthy; but it costs five francs (practically \$1.00) per liter. This price is prohibitive for many people, and the milk is difficult to obtain in this country. Goat's milk, we believe, offers more promise. We have seen several babies successfully reared upon it, after it seemed impossible for them to thrive upon cow's milk. Its high fat (4.09 per cent.) and proteids (3.69 per cent.) percentages make modifications of the milk necessary for some babies. Nevertheless, in the Island of Cuba, signal success is often obtained by permitting the baby to suckle directly from the goat. For some time it has been our intention to try some experiments of this nature.

But leaving these more or less theoretic considerations aside, there is only one animal that can begin to supply the enormous amount of milk needed for hand-fed infants, and that animal is the cow. As we have indicated, however, cow's milk is not only a highly infected article, but it differs materially in its chemical composition from mother's

milk. Ordinarily, cow's milk must be modified in some way or another in order that young infants may digest it. This naturally leads us to inquire what is the best method for modifying it. To most pediatricians in this country the answer to this question is found in "percentage feeding," guarded by knowledge of the baby's caloric needs (the calorimetric method). The percentage method of feeding has been woefully misunderstood by many of its critics. Probably, we may make the true method most clear by briefly tracing the growth of this "American Idea."

The first serious attempt to make a mixture of top milk, lime water and milk sugar, which should really simulate the composition of mother's milk ("humanized milk") was made by Dr. Arthur V. Meigs in 1882. Eight years later, Rotch of Boston, gave birth to the true "percentage idea" as we understand it, or should understand it, at the present time. This idea contemplated supplying the individual baby with the bulk of nourishment, and the percentages of fats, carbohydronates, and proteids, required for his proper nutrition and suited to his digestive power. Those critics who misapprehend percentage methods see but an ocean of complex formulæ, through which only an expert may successfully steer his course.

In 1891, in response to the efforts of Rotch, the first "Milk Laboratory" was established in Boston by the Walker-Gordon Company. Since then, the same company has established various model farms and various milk laboratories throughout the country. By Rotch, Holt and others, this is considered an ideal method of infant feeding.

The great advantages of the laboratory method are: 1. Flexibility in prescribing. One may write for practically any combination of proteids (casein or whey proteids), fats or sugars. He may prescribe any diluent desired. He may have the formula peptonized, pasteurized or sterilized. 2. Accuracy in compounding the various percentage mixtures. 3. Convenience. The milk comes to the consumer in individual feeding bottles, every one containing the prescribed amount for a feeding; and all that remains to be done in the home is to place the bottles in the ice chest, and to warm every successive bottle to the proper temperature at every feeding-time.

The prescription on page 124 will serve as an example.

The principal disadvantage of the laboratory method is its expense. Another disadvantage may arise because of the distance of the laboratory from the patient. True, the milk may be well iced and sent for considerable distances, but this adds considerably to the expense. In

R	Per cent.	Number of feedings, 7
Fats.....	3.00	.....
Carbohydrates, lactose (milk sugar) ..	6.00	Amount at every feeding 4 oz.
Dextrinize .....	.....	Other diluent
		Barley water 50 per cent.
Proteids..... { Whey.....	.75	.....
	.75	.....
{ Casein.....		
Total.....	1.50	Heat at .....
Peptonize.....	.....	Raw .....
		Yes
Sodium citrate—		
Per cent of milk and cream.....	.....	.....
Per cent. of mixtures.....	.....	.....
Sodium bicarbonate—		
Per cent. of milk and cream.....	.....	.....
Per cent. of total mixtures.....	.....	.....
Lactic acid bacillus.....	.....	.....

full justice to the laboratory feeding, it must be said that when very small gradations in the various percentages are necessary for success in feeding a given infant, the laboratory milk method offers the best solution of the problem.

**Home Modification of Milk.**—Westcott deserves credit for devising the first accurate formulæ for attaining percentage modification of milk and cream in the home. Later, he went several steps further, and in a masterful piece of original work furnished formulæ for whey, cream, whey-milk and cream, and other percentage modifications. We shall not do full justice to him or his methods in these pages, for, as we have indicated, it is our belief that this whole subject must be put before the profession in its simplest form. Otherwise it fails to elicit the interest that it deserves. H. C. Wood was in the habit of saying to his students: "I do not wish to bring you into a fog; but rather to bring before you a crystalline drop of dew." It shall be our object to crystallize this important subject of percentage feeding into such form.

Before describing two simple methods for the home modification of milk, we shall briefly outline a few principles that seem to constitute a necessary preamble:

*First.*—*How much* shall we give at a feeding? Holt has given us a very important rule here, though it is by no means a law: Add the figure 1 to the baby's age in months, and we shall derive the number of ounces he should receive at a feeding. Thus a baby at birth gets

1 ounce at a feeding; at one month he receives 2 ounces, and so on. Beyond six months of age we must usually proceed more slowly, and relatively few babies take more than 8 ounces at a feeding during the first year. Now, we have said that this is only a rule, for individual babies differ. Nevertheless, it is a safe rule, and would act as a valuable check upon the reckless methods of increasing food that we often see practised. A knowledge of the size of the infant's stomach at various ages is of course of great value.

*Second.*—*How often* should the baby be fed? A few years ago, this question would have received a stereotyped answer: "Every two hours until he is six weeks old; every two and one-half hours from then until he is three or four months old; every three hours from then on." These are still good rules for many babies; much better at any rate than the pernicious one which directs two-hour feeding without informing the mother of the necessity of lengthening the interval later. But it has been found that sturdy babies often do best on a three-hour interval from the first; and that later this may be increased to three and one-half or even four hours. Personally, we prefer, in general, the three-hour interval; but at six or seven months of age, we strive to have the baby sleep from 7 P.M. until 6 or 7 A.M. He thus secures the same number of feedings that he would secure if fed on the four-hour interval, and given one bottle at the parents bed-time.

But let us again recall, we are feeding individuals.

*Third.*—*How may* we think in percentages? If the profession at large would only realize how easy this is! Percentage feeding is intensely individualistic. It strives to attain a relative accuracy to supplant the hap-hazard methods of the past. There is nothing difficult, much less occult about it. One simply strives to find out the relative amounts of fats, carbohydrates and proteids, fitted to the individual babies nutritive requirements and digestive abilities. Except in the subjoined table, we shall not give percentage formula for various ages. We are striving to feed successfully individual babies. Holt, Chapin, and others have laid down some very important generalizations, however, and they may well serve for our guidance.

In the first three months of life, many babies do well upon formulæ in which fats and proteids bear the percentage relations of 3 to 1. (Thus we may have a 1.50-6-.50 or 2.25-6-.75 or a 3-6-1 formula.) From then, until the baby is somewhere between six and eight months of age, we often find most valuable formulæ with twice as much fat as protein. After this period most babies may take whole milk.

But these are only generalizations. Some babies cannot take fats

at all; some require more proteids than fat. Again, we repeat, we are striving to succeed with individual babies.

There is another aspect to percentage feeding: If the individual baby cannot digest the amounts of fats, carbohydrates or proteid that he seems to need, knowledge gained in percentage feeding furnishes us with certain food ideals, toward which we gradually and carefully try to proceed. It is here that the calorimetric method may prove a very valuable adjunct of the percentage method. Within the last few years, there has been a decided tendency to give less fat than in former years. We feel with Jacobi that this is possibly carried too far. Thus, he records the case of a baby who would not gain until he received  $2\frac{1}{2}$  per cent. of fat. On the other hand, we rarely prescribe more than 3 per cent. of fat in milk formula, and almost never more than  $3\frac{1}{2}$  per cent.

The following table (modified from Holt) summarizes the points just dwelt upon:

RULES (NOT LAWS) FOR GUIDANCE IN PERCENTAGE FEEDING  
(MODIFIED AFTER HOLT)

Age of the baby	Feeding interval	Night feeding	Amount at a feeding	Number of bottles in 24 hrs.	Amount in 24 hours	Percentage formula
Premature.....	1-1½ hrs..	3	1 dr. to ½ oz.	15-18	This is readily calculated by multiplying the number of ounces given at a feeding by the number of feedings in 24 hours.	Fat-lactose-protein .75%-4%-.25
First week.....	2 hrs. ....	1 Or 2	1-1½ oz....	9-10		1.00-5- .33
Second to fourth week.	2 or 2½ hrs.	1	1½ to 2 oz.	8-9		1.50-5- .50 1.88-6- .63 2.25-6- .75 3.00-6-1.00
Fifth and sixth week.	2 or 2½ hrs.	1	2-3 oz....	8-9		3.00-6-1.00
Sixth week to three or four months.	2½-3 hrs..	1	3-5 oz....	8		3.00-6-1.00 3.00-6-1.25 3.00-6-1.50
Four months to eight months.	3 hrs. ....	1	5-8 oz....	6 or 7		3.00-6-1.50 3.50-6-1.75 3.50-6-2.00
Eight to twelve months.	3-3½ or 4 hrs.	None...	8 oz. or more.	5		3.50-6-2.25 3.50-6-2.50 3.50-6-2.75 3.50-6-3.00 3.50-6-3.25 4.00-6-3.50

Many babies do better on a three-hour interval from the first. Some babies cannot take over 3 per cent. of fat. Still others must have fat-free milk or buttermilk.

But, let us return to the problem of home modifications: How are we going to modify milk or milk and cream so as to obtain these various percentage formulæ? Some have found a simple device like the "Materna" of great value; but in our own experience, two methods of procedure have proved most useful and most flexible. The first we may style the *milk and cream method*; the other may be termed the *top-milk method*.

**The Milk and Cream Method.**—In 1898 Baner of New York devised a very simple method of obtaining percentage modifications of milk and cream. It is really a simplification of Westcott's method. He strove to eliminate fractions by assuming that good herd milk contained 4 per cent. of fat; 4 per cent. of protein and 4 per cent. of lactose. Westcott practically accepts this elimination of fractions in his ingenious little "clock" or wheel device.

To work out a percentage formula by the Baner method, one has only to know the amount of food to be given in twenty-four hours, and the desired percentages of fat, lactose and protein.

$C$  = Cream.

$M$  = Milk.

$Q$  = quantity in ounces to be given in twenty-four hours.

$F$  = desired percentage of fat.

$L$  = desired percentage of lactose.

$P$  = desired percentage of protein.

Then:

$$\text{Cream} = \frac{Q}{12} \times (F - P)$$

$$\text{Milk} = \left( \frac{Q}{4} \times P \right) - C =$$

$$\text{Milk sugar} = \frac{Q \times (L - P)}{100} =$$

Diluent (water or other)

$$\text{Water} = Q - (M + C).$$

The factor 12 is used when a 16 per cent. cream is employed (16 per cent. fat—4 per cent. proteid). If a 12 per cent. cream is used, the factor is 8; if a 20 per cent. cream, the factor is 16.

The factor 4 is the percentage of protein in milk and cream. (This is really too high; but the error is on the safe side.)

Let us take a concrete example: To a baby three months old, we have decided to give eight feedings a day. Each feeding is to furnish 4 ounces. The amount given in twenty-four hours will therefore be 32 ounces. The percentage formula that we wish to furnish is  $F = 3.00$  per cent.;  $L = 6$  per cent. and  $P = 1.50$  per cent.

Then:

$$C = \frac{Q}{12} \times (F - P), \text{ or } \frac{32}{12} \times (3 - 1 \frac{1}{2}) = 4 \text{ ounces.}$$

$$M = \left( \frac{Q}{4} \times P \right) - C, \text{ or } \left( \frac{32}{4} \times \frac{3}{2} \right) - 4 \text{ ounces} = 8 \text{ ounces.}$$

(The cream is subtracted because it also contains proteid.)

$$L = \frac{Q \times (L - P)}{100}, \text{ or } \frac{32 \times (6 - 1 \frac{1}{2})}{100} = 1.44 \text{ ounces.}$$

(1 1/2 ounces would be near enough.)

$$\text{Water} = Q - (M + C), \text{ or } 32 - (8 + 4) = 20 \text{ ounces.}$$

If desirable, one may use lime water (*L.W.*) (5 per cent. — 10 per cent.) and barley water (*B.W.*) (50 per cent.).

Then:

$$\text{Water} = Q - (M + C + L.W. + B.W.), \text{ or } 32 \text{ ounces} - (8 + 4 + 1 \frac{1}{2} + 16) = 2 \frac{1}{2} \text{ ounces.}$$

The mother or nurse is instructed to mix (in a clean vessel): 4 ounces of certified cream (16 per cent.); 8 ounces of certified milk (4 per cent.); 1 1/2 ounces of lime water; 16 ounces of barley water, and 2 1/2 ounces of cool boiled water. She is further told to place 4 ounces of this mixture in every one of the eight bottles; to stopper these with sterile non-absorbent cotton, or rubber corks (that have been boiled), and to place all of the bottles on ice.

As we have stated previously, this method of modification is simple, flexible and useful.

**The Top Milk Method.**—Chapin probably deserves most credit for introducing this useful method of home modification; though many others have followed him in originating procedures of varying complexities and detail. Both Chapin and Holt teach that most babies, previously well, may be fed upon three sets of formula, viz.: With a ratio of fat to protein of 3 to 1; a ratio of 2 to 1; or a ratio of 1 to 1 (8 to 7 according to Holt). In our experience, this truth has been amply verified. These three sets of formulæ may be derived by

diluting milk from three levels of a quart jar; the upper third; the upper half, and the whole milk:

"Nor need we rack the weary brain,  
Nor search for method far:  
The good percentage end we gain  
From three levels of the jar."

The two writers differ somewhat in certain details: Thus Chapin's upper third is the upper 9 ounces ( $F:P::12:4$ ); Holt's, the upper 11 ounces ( $F:P::10:3.30$ ). In the milk from the upper half of the jar, Chapin finds 8 per cent. of fat and 4 per cent. of proteid; Holt 7 per cent. and 3.5 per cent. In the whole milk (rich milk), Chapin finds 4 per cent. fat and 4 per cent. protein; Holt, 4 per cent. and 3.5 per cent.

With the idea of popularizing these efficient methods, and presenting them to the profession in a simple way, one of us devised what he believes to be the simplest method of home modification that has been advanced. It is stated in two rules:

*Rule 1.*—Take the desired ratio of protein to fat and make a fraction of it ( $P$ =the numerator;  $F$ =the denominator); the resulting fraction represents the level of the jar from which the top milk (or whole milk) is withdrawn. Thus  $P:F::1:3$ : the top milk used is from the upper third.  $P:F::1:2$ : the top milk is taken from the upper half of the jar.  $P:F::1:1$ : (or 8:7 after Holt); the milk used is the whole milk.

*Rule 2.*—Make the desired percentage of protein the numerator of a fraction, and the percentage present in the milk the denominator. Multiply the number of ounces to be given in twenty-four hours by this fraction, and the result will be the amount of top milk or whole milk required. The remainder of the total amount will be the diluent (5 per cent. sugar of milk solution, etc.).

Let us take the same example that was used to illustrate the Baner method:  $Q=32$  ounces;  $F=3$  per cent.;  $L=6$  per cent.;  $P=1.50$  per cent.

*Rule 1.*—The desired ratio of protein to fat is 1 to 2; therefore the top milk is obtained by dipping off the upper one-half of the contents of a quart jar.

*Rule 2.*—The desired percentage of protein is  $1\frac{1}{2}$  per cent. The amount of protein assumed to be present in the milk is 4 per cent. (3.50 per cent., if we follow Holt). Therefore

$$\frac{32 \text{ oz.}}{1} \times \frac{1.50}{4.00} = 12 \text{ ounces of this top milk.}$$

Diluent (water, etc.) 32 ounces — 12 ounces = 20 ounces.

It will be observed that the 12 ounces of top milk is exactly equal to the cream (4 ounces) plus the milk (8 ounces) obtained by Baner's method.

The only directions given the nurse or the mother are that she shall dip off 16 ounces from the top of a quart jar of fresh milk (after standing four hours in the ice chest), shall mix this mixture thoroughly in a clean vessel and shall use 12 ounces of it in her mixture. Chapin's dipper, which holds just 1 ounce, is the best device for dipping off the top milk.

If the student will simply divest his mind of preconceived ideas concerning the difficulties of modification, and will work out a few hypothetic formulæ by these two methods, he will find that they have become his for all time. Then he will have attained more than a mere feat of memorizing; he will have acquired two useful methods of achieving percentage modifications that are far more efficient and reliable than any haphazard methods of the past or present.

**The Calorimetric Method.**—Through the laborious and epoch making endeavors of a number of German authorities, notably Heubner, Rubner, Cammerer, Söldner, Czerny, etc., we now know what a baby's energy requirements are. Up to six months of age, the infant's food should furnish 100 calories of energy for every kilo of his body weight. In the second half of the first year, this energy requirement averages 80 calories per kilo.

We use the calorimetric method in its application to infant feeding; but we use it principally as a check upon percentage methods. In the literature the method has been criticised from a number of standpoints; but there are two criticisms which have appeared to us most just. 1. The calorimetric method strives to prescribe so much energy for so many kilos of baby. This rather precludes such individuality in prescribing as the percentage method permits. Kerley states that he has tried to feed babies by this method; but that they have still remained hungry and that their parents have objected. 2. The same number of calories may be yielded by a number of different percentage combinations of fats, carbohydrates and proteids; yet one mixture may agree perfectly with a given baby, while another combination may result in untold disturbance of his digestion.

Let us apply it as a check to the percentage formula deduced by the two methods of home modification.

By Baner's method we derive the following mixture: Cream = 4 ounces, milk = 8 ounces, milk sugar = 1 1/2 ounces.

According to Brenneman an ounce of cream yields 54 calories of energy. An ounce of milk = 21 calories.

Then

4 ounces of cream  $\times$  54 calories = 216 calories.

8 ounces of milk  $\times$  21 calories = 168 calories.

1 1/2 ounces of milk sugar = 42.5 grams.

42.5 grams  $\times$  4.1 calories = 174.25.

From cream = 216 calories.

From milk = 168 calories.

From milk sugar = 174.25 calories.

Total = 558.25.

Now let us assume that the three-months old baby for whom this milk mixture is prescribed, weighs 11 pounds. A kilo is equal to 2.2 pounds, and the baby's weight expressed in the metric system is 5 kilos. His theoretic requirements are therefore 500 calories. It will be seen that this corresponds rather closely to the figures secured above. If there were evidences of over-feeding the mixture could very readily be lowered to the required calorimetric standard. As we have stated, we find this method most important in determining whether babies are receiving too much or too little energy from their percentage formulæ.

We append a few calorimetric values that will prove of service to the students in these estimations.

1 gram of fat yields.....	9.3 calories.
1 gram of protein yields.....	4.1 calories.
1 gram of carbohydrate yields.....	4.1 calories.
1 ounce of milk (Brennemann).....	21.0 calories.
1 ounce of cream (Brennemann).....	54.0 calories.

In a very recent article Holt makes some important contributions to such a list:

#### FROM 4 PER CENT. MILK

1 ounce of 7 per cent. milk (upper 16 ounces).....	27.5 calories.
1 ounce of 6 per cent. milk (upper 20 ounces).....	25.0 calories.
1 ounce of 5 per cent. milk (upper 24 ounces).....	22.5 calories.
1 ounce of 4 per cent. milk (whole milk).....	20.0 calories.
1 ounce of 3 per cent. milk (after skimming off 2 ounces) ..	17.5 calories.
1 ounce of 2 per cent. milk (after skimming off 4 ounces) ..	15.0 calories.
1 ounce of 1 per cent. milk (after skimming off 8 ounces) ..	12.5 calories.
1 ounce fat-free milk.....	10.0 calories.
1 ounce whey.....	10.0 calories.
1 ounce milk sugar (weight).....	116.0 calories.
1 ounce milk sugar (volume).....	72.0 calories.
1 even tablespoonful of milk sugar.....	44.0 calories.
1 ounce barley flour (by weight).....	100.0 calories

1 ounce barley water (tablespoonful to the pint).....	2.0 calories.
1 ounce malt soup extract.....	80.0 calories.
1 ounce condensed milk.....	132.0 calories.
1 ounce olive oil (by volume).....	245.0 calories.

**The Feeding of Difficult Cases.**—The rules laid down in this discussion will serve very well to direct us in the feeding of babies whose stomachs and bowels are not disturbed by improper feeding. But what of the babies whose digestive organs are disturbed? What of the infants who have anatomic lesions of the alimentary tract? What of those who are suffering with gastro-enteric infection? How shall we deal with the atrophic baby? These are questions of great import—too grave to be answered by nurses or mothers. They are puzzling questions, often making the general practitioner despair, and usually meaning for the specialist a long, arduous and carefully planned campaign. To wage his battles successfully, the pediatrician must elicit careful histories; must study the patient intelligently, must observe stools, vomitus and other excretions; must sometimes invoke the aid of the laboratory, and must possess resourcefulness in meeting the various pathologic conditions that arise. We shall try to elucidate the various principles that guide him in his studies and direct him in his therapeutic efforts, when such feeding cases come under his care.

A. The careful elicitation of the history: Holt lays great stress upon this matter and such investigation may be regarded as a prerequisite of clinical success. In general it is a good plan to find out every food that has been given the baby. Then proceed to find out how every one of these foods have affected the baby's stomach, his bowels and his nutrition. We cannot go into these matters in detail; but the appended table (page 133) has proved of inestimable service to us in interpreting clinical phenomena, and in teaching medical students.

It is not always possible to detect the faulty element in the diet until several or more percentage formulæ have been employed. Sometimes more than one organic constituent may play a part in the production of the disturbance. Thus the fat and proteid percentages may both be too high, etc.

B. The initial formulæ employed in feeding cases should contain low percentages of the organic constituents, particularly of protein and fats. Holt avers that there is no greater mistake in infant feeding than to "start high." The infant should not be kept indefinitely upon these low percentage mixtures, however. Their strength should be increased as the baby's digestive powers warrant such changes. One

## SYMPTOMS THAT GOVERN US IN THE PERCENTAGE FEEDING OF THE INDIVIDUAL BABY

Symptoms	Proteid	Fat	Carbohydrate <sup>1</sup>
of Excess	<p><i>Intestinal colic.</i> The baby is often relieved by the passage of gas from the bowel.</p> <p><i>The stools</i> are often green, and there may be constipation or diarrhea. In the former case, there may be a putrefactive odor. Curds may also be present, but these are hard to tell from masses of fat or soap.</p> <p><i>Vomiting</i>, when present, is liable to appear soon after feeding, and the vomitus contains curds.</p> <p>These babies may gain rapidly in weight despite their discomfort.</p>	<p><i>Vomiting</i> often occurs hours after feeding, and usually the odor of butyric acid is very apparent.</p> <p><i>The stools</i> are often loose, and are also sour (butyric acid). They are frequently very yellow (gelbfärbung-Biedert's fat diarrhea). They also contain fatty masses. On the other hand, there may be constipation, when the stools are peculiarly light in color and dry.</p> <p><i>Colic</i>, when present, is usually gastric (Westcott).</p>	<p><i>Vomiting</i> may be very frequent and the vomitus very sour. (This is due to lactic acid, however.)</p> <p><i>The stools</i> are acid, and often excoriate the buttocks. They are often green.</p> <p><i>Fretfulness</i> is liable to be present most of the time.</p> <p>Rickets (?). In reality, this may result because the excess of carbohydrate is used to atone for the deficiencies of other organic elements.</p> <p><i>Scurvy.</i></p>
of Deficiency in	<p>Hunger.</p> <p>Failure to gain weight.</p> <p>Rickets.</p> <p>Scurvy.</p>	<p>Rickets.</p> <p>Hunger and failure to gain weight.</p>	<p>Not definitely known; but it seems rational to use an amount of milk sugar equivalent to that which nature has placed in mother's milk.</p> <p>In certain states (diabetes, etc.) disaster results when we give too little carbohydrate.</p>

should never be satisfied long with milk formulæ containing less than 1 per cent. of the protein element. (See rickets and scurvy.)

C. Carbohydrate diluents are useful where difficulty is experienced in the digestion of casein. Jacobi strongly advocated such diluents years ago. Chapin has also been a powerful advocate of the use of such diluents. The latter authority uses "Cereo," or some other good thick malt extract to transform the starch in barley water, etc., into dextrin and maltose. A dram of such an extract is added to a pint of the cereal decoction after the latter has been boiled and has been cooled to a point where it can be tasted.

Whatever academic discussions may arise concerning the baby's

<sup>1</sup> Note.—Finkelstein and his pupils believe that milk sugar and cane sugar cause the initial harm in the digestive and metabolic disturbances of infancy. They also believe that bacteria play secondary rôles in gastroenteric infections.

ability or inability to digest starch, we agree most cordially with these authorities that many babies can digest higher protein percentages when cereal diluents are employed in their milk mixtures. In the light of recent investigations, it is highly probable that the carbohydrate exerts more than a beneficial mechanical action on curd formation in the stomach. It is our custom to employ 50 per cent. of barley water or other cereal decoction, in most of our milk formulæ. Thus if we are prescribing a total quantity of 32 ounces of liquid food for the day's consumption, we usually employ 16 ounces of barley water or "Cereo" barley solution in making up the mixture.

*D. The Use of Whey and Whey and Cream Mixtures:* Some babies who cannot digest mixtures containing whole milk or milk and cream, can be fed upon whey and cream mixtures. This clinical fact would seem to furnish a refutation of the teaching that casein never disturbs the infantile digestion. The only difficulty encountered in the use of such mixtures is that of securing a sufficient percentage of the protein element. As we have mentioned, Westcott has furnished ingenious formulæ for achieving percentage modifications in the use of whey and cream mixtures. It is our custom to employ a very simple method of modification: Fifty per cent. volume of our mixture is contributed by the whey. This furnishes approximately .50 per cent. of protein, and 2.50 per cent. of lactose. The small percentage of fat in whey we disregard in the calculation. Fat, lactose, and some protein are furnished by the cream. These percentages we calculate. It is assumed that certified cream contains 16 per cent. fat, 4 per cent. lactose and 4 per cent. protein (see Baner's formula). Now suppose we wish to use a formula containing 2 per cent. of fat. We also wish to give 24 ounces of food in the twenty-four hours. Then:

$$\text{Cream} = \frac{2\% (\text{desired percentage of fat})}{16\% (\text{percentage present in cream})} \times 24 \text{ ounces} = 3 \text{ ounces}$$

But if the cream furnishes 2 per cent. of fat, it also furnishes one-fourth as much lactose (.50 per cent.) and protein (.50). A mixture made up as follows:

Whey.....	12 ounces.
Cream, 16 per cent.....	3 ounces.
Boiled water (or other diluent) .....	9 ounces.

The mixture contains approximately 2 per cent. fat; 3 per cent. milk sugar (2.50-.50) and 1 per cent. protein (.50-.50). If we add 3 per cent. lactose (by weight) to this solution, we will have a 2, 6, 1 formula.

Whey is made as follows: A quart of fresh milk is placed in a clean bowl. To it is added one or two teaspoonfuls of "elixir pepsin." The milk is then placed over the bars of the stove (preferably kept at 100° F.) until a curd is formed (about half an hour). The curd is then broken up with a fork, and the whey is filtered through several layers of sterile cheesecloth spread over a broad surface. The whey should then be pasteurized at 140° F. for half an hour.

**E. Peptonization.**—In spite of recently expressed doubts concerning the value of artificial digestion in infant feeding, we still find that peptonization helps us over many a rough place. It has been stated that the young infant expends one-eighth to one-sixth of his available energy in the process of digestion. To save this much of the expenditure for the weak infant, may mean that we enable him to gain in weight, or that we actually save his life. We have come to regard peptonization as a useful measure under the following conditions: In prematurity; in partial substitute feeding, when the breast milk is high in solids, and the baby is suffering from protein indigestion; in cases where it is difficult to give a sufficient amount of proteid without such predigestion; in some cases of pylorospasm; in some cases of infantile atrophy; in the cases of weak infants during dentition periods, and in some cases in the second year of life, when the babies are able to take other food, but are still having difficulty in digesting milk.

Milk peptonized by the hot process is also sterilized, and this latter fact, while constituting a safeguard in some cases, still makes such a food objectionable for long continued use. This statement introduces us naturally to some criticisms of predigestion in infant feeding. Babies kept upon peptonized milk for a number of months may develop scurvy. Again, while the products of zymolysis may stimulate secretion of digestive juices up to a certain point, it is still true that the baby taking a peptonized food is not developing his own digestive organs as he should. We quite agree with Holt that digestive upsets may take place in babies who have been so fed for several months; and that under these conditions the babies may seem unable to digest anything at all. Still again, we have seen babies fed on peptonized formulæ reach a stage when they ceased to gain in weight. Substitution of a fresh milk formula, of exactly the same composition (minus the peptonization) has sometimes resulted in an immediate rapid gain in weight. Possibly such cases are really examples of incipient scorbutus.

**The Method of Peptonization (Partial Peptonization).**—It is the usual custom to employ 5 grains of pancreatin and 15 grains bicarbonate of soda to every pint of the milk or to every pint of the milk mixture. Some authorities peptonize the milk and then add it to the rest of the mixture, but recognizing that pancreatin contains proteolytic, steatolytic, diastatic, milk curdling and inversive (?) ferments, we usually advocate peptonization of the whole milk mixture. In giving directions to mothers and nurses, we divide the process into steps as follows: *First Step:* Bring the mixture of milk, cream, barley water, etc., to blood heat ( $100^{\circ}$ ) in the inner compartment of a double boiler. This attains a good temperature for digestion. *Second Step:* Remove the mixture from the fire, and place it aside on a table. Stir in the pancreatin (5 grains to the pint) and bicarbonate of soda (15 grains to a pint), or the contents of a peptonizing tube, or a measure of "peptogenic milk powder" (to every pint). Let the mixture remain off the fire for 10 minutes. (This precaution prevents too high a temperature.) During this period digestion proceeds. *Third Step:* Replace the mixture on the fire, and bring it to the simmering point as quickly as possible. This is done to destroy the ferments and prevent the formation of bitter products. The mixture may then be placed on ice.

**F. Sodium Citrate in Infant Feeding.**—Wright, Cotton, and others have secured good results with this salt in infants who seem unable to digest the casein of cow's milk. The addition of sodium citrate to bovine milk changes the calcium casein into a calcium citrate, thus delaying the coagulation of the casein. Most authorities who have used it have used from 1 to 3 grains in every ounce of milk mixture. Holt has found this salt useless in atrophic infants. He finds, however, that its employment enables one to give larger percentages of proteid without creating disturbance. We cannot speak from our own experience, having been deterred from using it by the fear of its possible purgative action.

**G. Buttermilk Feeding.**—Taken all in all, the use of buttermilk in infant feeding represents one of the most valuable additions to our modern dietetic armamentarium. It is really an old time method (in Holland); but has been placed upon a scientific basis only during the last decade.

There are probably three reasons why buttermilk acts as beneficially as it does: 1. In buttermilk the calcium casein is changed into a casein lactate. This is very readily digested and absorbed. We are thus able to give the tissue builder in high percentages. 2. The fat in a

properly made buttermilk is reduced to a low percentage. Thus we have a frequent cause of infantile disturbance removed. 3. As ordinarily employed, in buttermilk mixtures, the readily available carbohydrates have been added to atone for the deficiency in fats.

Care should be taken not to confound soured milk, still containing its butter-fats, with true buttermilk, as milk dealers so often do. If a fresh buttermilk cannot be secured daily from a reliable source, it should be made in the home as follows: A quart of fresh milk is placed in a sterile pitcher. One of the good commercial acidifying tablets, preferably containing the Bulgarian-bacillus, is then crumbled into the milk. The pitcher is covered with a sterile towel, and the mixture is permitted to stand at room temperature for from twenty-four to thirty-six hours. At the expiration of this time, the soured milk is churned. (A small glass churn, containing just a quart is convenient for household use.) When the butter fat is removed by churning, buttermilk remains.

Good buttermilk should contain from 2 1/2 to 3 1/2 per cent. of proteid and from .25 to .50 of fat.

As has been stated, buttermilk is usually modified for infant feeding. A very useful formula was suggested by de Mattos; but his original mixture probably contained too much sugar (60-90 grams to the liter). We usually prepare the buttermilk mixture as follows:

To a small amount of buttermilk is gradually added one level tablespoonful of cane sugar and two level teaspoonfuls of wheat flour, barley meal or some prepared infant food. When this mixture has been made into a smooth paste (creamed), it is stirred into the remainder of a quart of buttermilk. The mixture is then placed in an appropriate vessel, and is gradually brought to the simmering point, with constant stirring. Indeed though de Mattos suggested letting the mixture "boil up three times" it is often advisable to heat it to a lower temperature than the simmering point. In this way we may avoid the occurrence of "lumping" in the mixture.

Buttermilk mixtures, such as the above, may be given in full strength, or they may be diluted to fit the infant's digestive capacity. We may also pursue either percentage or calorimetric methods of feeding.

*Indications for buttermilk feeding:* The first of these is *fat indigestion*. In *atrophy*, it may act almost as "a specific." We have also come to employ it after the cessation of gastro-enteric infections. Only twice have we seen babies disturbed by this last named employment, as they are so often by the return to the use of sweet milk mixtures.

Some of the most remarkable gains we have observed in hand-

fed babies have occurred in atrophic infants fed upon buttermilk. One such baby, failing to gain on the mother's milk, failing also to gain upon the milks of two wet nurses, gained a pound and seven ounces in a single week on buttermilk.

Most authorities agree that buttermilk mixtures should not be given over a considerable period of time. Rickets often occurs in babies so fed. Again babies will often gain wonderfully for several weeks, and will then cease advancing until fats are added to their food. Vomiting has not always proved a contraindication in our experience. Indeed, four of our cases of pyloric stenosis have done well on buttermilk mixtures. It is conceivable that scurvy might result from the long continued use of such a food; but we know of no such cases.

Buco-buttermilk is an excellent commercial product which has yielded splendid results in some of our cases.

**H. Temporary Withdrawal of Milk in all Acute Cases of Gastro-intestinal or Metabolic Disturbances.**—To the pediatrician's mind, this is an expedient that immediately suggests itself under such conditions. The "starvation day" is the first therapeutic measure which he directs. Very often, indeed, the day of abstinence from nourishment must be stretched into days. In the presence of a gastro-enteric infection of the summer season, we rarely return to milk feeding under a week's time. Even then, experience teaches that we must proceed very slowly. Usually in the presence of such infectious conditions, we give only boiled water (often cool) on the first day of the illness. Barley water is given upon the second day. On the third day the barley water is sweetened. Then for the remainder of the week, we use a mixture containing equal parts of albumin water and dextrinized barley jelly. Milk, when we return to it is given in a very diluted mixture, and is peptonized; or else we employ a diluted buttermilk mixture (see above).

Finkelstein and his followers, regard milk sugar, or cane sugar as the initial disturbing element in the food. He contends that bacteria and fats play etiologic roles only after sugar has first disturbed. He thinks that casein is nearly always well digested.

In cases of indigestion and intestinal decomposition, he and Meyer employ albumin milk ("eiweiss milch"). This is prepared in the following manner:

One liter of milk is treated with 1 dram of liquid rennet. This is then permitted to stand in a water bath for half an hour. It is kept at a temperature of 107.6°. At the expiration of this time, the curd (casein and fat) is placed in a linen bag, and the whey allowed to filter

off for one-half hour. The remaining curd is then mixed with one-half a liter of water, and is twice worked through a hair-sieve. One-half liter of buttermilk is then added. The resulting mixture is said to contain fat 2.5 per cent., sugar 1.5 per cent., protein 3 per cent., and ash .5 per cent.

Albumin milk should be fed in very small quantities at first ( $\frac{1}{2}$  ounce to 1 ounce to a feeding), and the amount gradually increased. As a rule, babies do not gain upon it until sugar is added, and this should be in the form of maltose or "dextrin-maltose." ("Nahr-zucker.")

Finkelstein's methods have not been generally employed in this country, and reports differ concerning their efficiency. Students returning from abroad are enthusiastic; but a recent report of Morse fails to show the brilliant results anticipated.

I. Wet nursing is sometimes the only measure that will bring success. This fact has been considered in its appropriate place.

J. *Proprietary Infant Foods*.—If the careful elicitation of the baby's dietic history reveals that some infant food has agreed with him better than other foods, such a statement should not be ignored. One may continue to advise such a food as a carbohydrate diluent; or if there is an acute disturbance, one may depend upon it solely until the disturbance ceases. This statement truly portrays the principal uses of proprietary foods in infant feeding. Sole dependance upon them for a length of time too often means eventual scurvy, or rickets, or both. It results too often in impaired digestion. We repeat, proprietary infant foods are not to be recommended, unless as carbohydrate diluents of milk, or in disturbed gastro-intestinal conditions, when milk cannot be taken safely. A minor use of them—one to which we frequently resort—is for infants journeying by train or steamer. Condensed milk is very satisfactory for this purpose. Residence in the tropics, may again occasion the temporary employment of such foods.

If infant foods are used as carbohydrates in the milk mixture, they should be employed as accurately as we employ lactose or barley. For this purpose, one should possess a knowledge of the compositions of several of the standard foods:

Chapin classified the various proprietary infant foods in a very instructive manner, and the following tables are principally derived from his work upon "The Theory and Practice of Infant Feeding":

CONDENSED MILKS AND CREAM;<sup>1</sup> UNSWEETENED CONDENSED MILK; WHOLE MILK (SO-CALLED EVAPORATED CREAM)

Water	Fat	Proteid	Milk sugar	Solids not fats	Cane sugar	Ash	Brand
68.27	10.10	7.36	11.03	.....	.....	1.85	"Ideal"

Evidently Skimmed or Partly Skimmed Milk

74.29	1.80	8.97	.....	23.91	.....	2.39	"Monarch"
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Sweetened Condensed Milk—Whole Milk

28.41	8.44	7.23	11.69	41.52	.....	1.80	"Eagle"
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Evidently Skimmed or Partly Skimmed Milk

25.68	.71	10.35	16.85	.....	43.09	2.48	"Cowslip"
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Condensed Cream

59.60	34.19	.....	.....	6.21	.....	0.53	"Dahl's Gold Medal."
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OTHER PROPRIETARY INFANT FOODS

"Class I. To be used with water; no cooking required."

Class I	Moisture	Fat	Proteid	Soluble carbohydrates	Insoluble carbohydrates	Ash
Allenburg's food No. 1...	5.7	14.00	9.7	66.85	.....	3.75
Allenburg's food No. 2...	3.9	12.30	9.2	72.10	.....	3.50
Malted milk.....	2.55	1.41	14.00	63.87	15.68	3.57

Cereal milk and milking are other examples of this class. All are prepared by adding dextrin and maltose derived from wheat flour to milk. The mixture is then evaporated to dryness.

<sup>1</sup>Analyses furnished Dr. Chapin by Major Alford. Examples of the various classes are here quoted.

## Class II. "To be used with water; cooking required."

	Moisture	Fat	Proteid	Soluble carbohy- drates	Insoluble carbohy- drates	Ash
Nestle's food.....	2.18	4.45	10.72	43.48	35.34	1.60

Prepared by adding to dried milk, sugar and baked wheat flour.

## Class III. "To be used with milk; no cooking required."

	Moisture	Fat	Proteid	Soluble carbohy- drates	Insoluble carbohy- drates	Ash
Mellin's food.....	4.72	.30	10.10	82.06	.....	3.50

Such foods are made from wheat flour by converting the starch into dextrin and maltose by means of a diastatic ferment.

## Class IV. "To be used with milk; cooking required."

	Moisture	Fat	Proteid	Soluble carbohy- drates	Insoluble carbohy- drates	Ash
Carnricks soluble food...	5.69	2.18	16.60	38.21	34.54	2.78
Imperial Granum.....	6.04	.72	13.77	3.94	67.46	.49
Ridge's food.....	8.12	.48	13.83	5.02	69.24	.53
Health Food Co.'s barley.	10.92	.89	5.82	?	80.35	.86
Robinson's patent barley.	9.41	.41	7.46	2.91	78.66	.94

"These are said to be made of baked wheat or barley flours."

Eskay's food, to which the white of egg is added, may be included in this group. Benger's food, much used in England, contains pan-creatin, and peptonizes the milk to which it is added.

	Moisture	Fat	Proteid	Soluble carbohy- drates	Insoluble carbohy- drates	Ash
Eskay's albuminized food <sup>1</sup>	2.70	1.16	5.82	67.81	21.21	1.30
Benger's food <sup>2</sup> .....	8.30	1.20	10.20		79.50	0.80

If one will compare the analyses here quoted with analyses from other sources, he will be almost forced to the conclusion that many of these foods do not have fixed chemical compositions; but that different samples at different times yield different results.

<sup>1</sup> From Holt's "Diseases of Infancy and Childhood."

<sup>2</sup> From Koplik's "Diseases of Infancy and Childhood."

**Diet During the Second Year of Life.**—While babies will not develop infantile scurvy when fed upon fresh milk containing proper proportions of organic solids, the antiscorbutic properties of milk are very slight. Some babies who have not gained rapidly, even with perfect digestion when fed on good milk formulæ, forge ahead rapidly when orange juice is added to their dietaries. It has become customary to give babies orange juice when they are six or seven months old. It is our custom to begin with fl. ʒ ij given three times daily midway between bottles. If no vomiting or looseness of the bowel results, we give the baby the strained juice of half an orange once a day. By the end of the first year, this has been increased to the juice of an orange. The “hoped-for” laxative influence of orange juice is not always realized. We wish it were.

Milk is also a poor iron-bearing food, and toward the middle of the first year the baby has exhausted much of his available supply of iron in the liver; it is therefore well to give some other food than milk beyond this period. This may be done by simply adding sufficient carbohydrate element to the milk. Ordinarily, we supplant our barley water in the milk mixture with a dextrinized barley jelly (Cereobarley of Chapin, etc.). Beef juice, we reserve for special indications: viz., anemia, scurvy, or rickets. If employed, we always filter it through four layers of cheese-cloth (Miller) to avoid infection of the baby with ova of the beef tape-worm.

At a year of age then, the baby is taking this milk mixture, the juice of an orange, and some good carbohydrate in his milk. At that age, he is usually placed upon a fuller dietary.

We generally give: *Upon arising*.—A bottle of milk (full strength or three-fourths strength, and containing a cereal).

*Breakfast* (soon after the morning bottle).—The juice of an orange, two level tablespoonfuls of a well-cooked cereal, with milk and salt or butter<sup>1</sup> and salt (no sugar). A half slice of buttered toast or zwieback.

10 A.M.—A bottle of milk (as before).

1 P.M.—Four to six ounces of beef, mutton or chicken broth with rice, barley or arrowroot thickening. Two level tablespoonfuls of boiled rice, with butter and salt. Toast, zwieback or crackers. A half cup of junket. (If the child is constipated, an ounce of prune juice in place of the junket may be given—Starr.)

4 P.M.—Milk.

7 P.M.—Milk.

<sup>1</sup> We never see bad results from butter, as we do from butter-fats in cream. We believe it to be an easily digested and highly nutritious substance..

Between fifteen and eighteen months of age, we add an egg to the diet at breakfast time. This may be taken with orange juice in the raw state, or it may be given in the customary "coddled" (or "curdled") form. We often alternate with the raw and "coddled" egg on successive mornings; but on the whole we regard the raw egg as more readily digestible and more nutritious. (Vitellin is destroyed by heat.) Mothers should be warned concerning possible infantile idiosyncrasies toward eggs. We have seen vomiting promptly follow the administration of the yolk of egg, though the baby took the white alone without disturbance. On the other hand we have observed the phenomena of anaphylaxis when the white of egg was eaten.

A mealy baked potato, with butter and salt, may be given in place of rice when the baby is eighteen months old. Potato disturbs some babies, however, even at this age. If well taken, we give potato one day, and rice upon the next. The methods of preparing some of the foods mentioned will now be given:

*Oat or Barley Jelly* (the former contains slightly more starch than the latter, while both contain a certain per cent. of fat).—Oat jelly can be made in the following manner: Four ounces (120 gm.) of coarse oatmeal are allowed to soak in a quart of cold water for twelve hours. The mixture is then boiled down so as to make a pint. It is then, while hot, strained through a fine cloth. Barley jelly is made in practically the same manner. We follow Chapin, and add a teaspoonful of "cereo" or other thick malt extract when the mixture is cool enough to taste. We thus produce a partial digestion of the starch (dextrin and maltose).

*Flour ball* is made as follows: A pint of wheat flour of good quality, without bran, is tied tightly in a pudding bag. This is placed in a saucepan of water and boiled constantly for ten hours. It is then allowed to cool, the bag removed, and the outer covering of dough cut away. The yellowish-white interior of the mass consists almost entirely of dextrin, which has been formed from the starch during the process of cooking. This interior is reduced to a powder by grating. To prepare for use in a nursing-bottle, rub a teaspoonful of the powder with a tablespoonful of milk until a smooth paste is formed; a second tablespoonful of milk is now added, with constant rubbing. This quantity should be poured into eight ounces of hot milk, the milk being continuously stirred while the paste is poured in. Flour ball is constipating, but is sometimes of considerable service as a carbohydrate diluent of milk after diarrheal diseases.

*Beef-juice* is prepared in the following manner: A pound of beef

sirloin should be warmed in a broiler before a quick fire; it should then be cut into small pieces and placed in a lemon-squeezer or meat-press, so as to express the juice, which is caught in a hot cup. All fat should be removed. Care should be taken not to cook the meat.

*Beef Broth.*—One pound of lean beef should be minced finely and put with its juice into an earthen vessel containing a pint of water at 85° F. (29.4° C.). It should then be allowed to stand for one hour, after which it is strained, preferably through stout muslin, until all the juice is removed from the meat. The liquid is then placed on the fire and heated slowly just to the boiling-point, being stirred all the time; it is then removed and seasoned with salt.

*Mutton broth* is prepared by gently boiling one pound of loin mutton in three pints of water until the meat is tender; a small quantity of salt should then be added, and the whole strained into a basin; skim off the fat as soon as cold. Both beef and mutton broths should be warmed before giving them to the child.

*Chicken broth* is made in the following manner: A small chicken, or half of a large one, after being thoroughly cleaned and having all the fat and skin removed, is chopped into small pieces, bones and all; a pinch of salt is added and the whole is placed in a saucepan containing a quart of boiling water. The cover of the saucepan should be closed tightly and the contents allowed to simmer over a slow fire for two hours. After removing, allow it to stand, still covered, for an hour. The broth is then strained through a sieve. From three to five ounces should be given at one feeding. As a general rule, broths should only be substituted for milk for about one feeding daily two or three times a week.

#### DIET BEYOND THE PERIOD OF INFANCY

We firmly believe that nature points out the period when the child should begin to eat flesh. That period is the completion of his primary dentition. When the second molars have erupted, therefore, we add rare beef or mutton to the menu, giving it in the middle of the day. (In tuberculous or rachitic children, it may be necessary to give pressed beef, etc., before this period.) Breakfast bacon may soon be added, and chicken may replace the red meats.

Green vegetables are of great service to children, but sometimes disturb the bowels, and very often they do not appeal to the palate of the child. Following Starr, we sometimes give spinach in the second year of life, but it often occasions looseness of the bowels, and sometimes appears quite unchanged in the stools. Beyond the period of in-

fancy, children may take lettuce, spinach, ochre, tips of asparagus, cauliflower, Brussel's sprouts, young lima beans, string beans (if well strung), etc. The great difficulty is that they usually fail to appeal to children. Contrary to usual teaching, we are in the habit of adding a modified French or mayonnaise dressing to these green vegetables. We style the results "green vegetables beatified." The dressings are made with lemon juice instead of vinegar, and contain no high seasonings. After all, when so made, they represent but emulsions of olive oil.

*Beverages.*—Aside from milk and water, cocoa is the only beverage that we permit through the whole of childhood. To deliberately give a growing child tea or coffee seems well nigh a wicked thing. National customs vary; but we feel equally strongly that no malt or alcoholic liquors should be given to children in health. Growing tissues do not need stimulation. In disease, tea and alcohol may both be important remedies.

*Desserts.*—Junket, puddings, custards, stewed fruits, baked apple, and ice-cream (once a week) represent desserts that most children may have. Ice-cream is best home made, and should contain simple, pure flavors.

*Fruits.*—Aside from the sub-acid fruits, particularly the orange, cooked fruits tend to agree with children better than raw ones. Apples, we never give until after the period of infancy. They often cause flatulence, and sometimes restless sleep. On the other hand, we must agree with the apparently heretical views of J. D. Milton-Miller concerning the value of the banana. The banana (really a yam) appeals to the child's appetite, and often stimulates him to take other food. Again, properly prepared, it rarely disagrees. We scrape the outside of the banana and then slice it in very fine transverse sections. It may then be eaten with milk or cream and sugar. Sliced bananas may also be given with sliced oranges or orange juice.

*Nuts.*—We are quite in accord with Dr. Wm. E. Robertson's views concerning the nutritive values of nuts. We do not give them to young children, because they are often hard to digest. In under-nourished older children, however, we often find them of great service. They are given at the end of the meal, and usually in the form of a nut sandwich (toast, lettuce, mayonnaise, and nuts ground up between the layers of lettuce). We shall not give a number of dietaries for different ages. We have tried rather to point out certain principles which guide us in the selection and preparation of food for children. Let us mention two more important guiding thoughts:

Do not coddle children in dietetic lines. Remember that food should do more than nourish. It should prepare a gastro-intestinal tract for the future exigencies of life.

Again, remember that diet represents only one phase of nutrition. The soldier on the march, or the camper in the woods, can eat almost any food with impunity. See that our children get sufficient exercise, rest, sunlight and fresh air to develop good digestions.

It is necessary, however, that we should mention some of the forbidden fruits of childhood: Cake, candy, substances that are known to cause indigestion, pastry, fried things (except breakfast bacon and fried mush, if done in deep fat), coffee and tea, malt and spirituous liquors, highly seasoned foods and dressings, shell fish (except raw oysters), fancy desserts, cabbage, most cheeses (unless for the tuberculous), and berries (for young children), are foods that should be denied. Concerning candy, we agree with Starr, that it should be ranked among the thousand and one other unattainables of childhood. The amount of suffering that children undergo because of their excessive fondness for sweets is wholly disproportionate to any pleasure they may derive from them. That parents will deliberately permit their children to disturb themselves to the extent that they often do at the Christmas and Easter seasons, seems to us nothing short of cruel. The same parents would probably exercise every caution to prevent the same children from burning themselves or suffering other physical injuries. To children with weak digestions or those with diabetic inheritance, such "sweet-sprees" may be fraught with actual danger. Still the candy habit is deeply engrafted on the public, and its indulgence is viewed with more or less complacency; so it may be best to allow a very small quantity of candy once a week, and to render the indulgence safe the confection is given on a full stomach (after the Sunday dinner). On the other hand, the child's need for sugar is fully recognized; but sugar should be given mixed with other food, not in concentrated form.

We have spoken of substances that are known to cause indigestion. This brings us into the realm of individual idiosyncrasy. Many a child will be disturbed by seemingly harmless articles of diet. If such relationships between cause and effect are well established, the child should have such disturbing elements eliminated from his diet.

A word about berries: In the craw of the bird, small stones and gravel may be ground to powder, but the berry seed not only traverses the bird's alimentary canal, but it actually grows where deposited upon the ground. Theoretically, such food seems improper for children, and practically berries often disturb. The popular straw-

berry is an offender in a number of different ways. We are in the habit of excluding berries from the child's menu until the subject is seven or eight years old. Eating between meals should always be frowned upon. Some children need more meals than others, but these can be given with perfect regularity. In giving meals regularly, we not only spare the child much digestive disturbance, but we also tend to inculcate good habits of eating which may prove of service to him all his life.

In difficult feeding cases in childhood, the calorimetric method may prove of much service.

We shall not devote special attention to "sick diets" in this section, as the question of diet is considered in the study of every disease in which feeding forms an essential part of treatment.

### WATER DRINKING IN INFANCY AND CHILDHOOD

Pure water should be given to infants from the very first. As many have taught, the proteins of colostrum and of breast milk may be so reduced in percentage. As stated, it is our custom to give the baby two drams of tepid water before every nursing, particularly when the infant is suffering from protein indigestion. The young baby thus receives between 2 and 3 ounces of water a day. Jacobi has claimed for this early administration of water another function of importance. The pelvis of the kidneys in the new-born often contain uric acid infarcts. Free administration of water, with its consequent free secretion of urine is of value in the removal of these small concretions.

As the baby grows older, the amount of water given him should be gradually increased. Many a baby who appears to cry from hunger really cries because he is thirsty. Particularly during warm weather, when his skin is freely active, free water drinking greatly safeguards the comfort and health of the baby.

Some babies do not seem to care for water, even when they have fever. Usually this apparent antipathy may be overcome by experimentation with different temperatures of the water administered. Some babies like hot water best, while others will take it with more avidity when the water given is below the tepid temperature. In general, the temperature of the drink may be decreased as the baby grows older.

Modern views concerning the administration of water to older children appear most heretical to the members of the last generation. It is not so very many years ago that children were denied water

drinking at meals. This seems very surprising to us when we think that the action of all enzymes is dependent upon hydrolysis. Far from forbidding water drinking at meals, we should encourage the habit in moderation. As a rule, indeed, it needs little encouragement. Most children should be allowed somewhere between one and two glasses of cool or even cold water at every meal. Though its use may prove harmful to infants, we are convinced that most children may drink ice-water with positive advantage to their appetites and digestions. Even babies may take it freely when suffering with febrile diseases. More than once, we have seen vomiting checked and life apparently saved by the free administration of ice-water to the infant suffering from gastro-enteritis with severe systemic intoxication.

A number of years ago, in experimenting with dogs with gastric fistulæ, Reichert found that ice-water markedly stimulated the secretion of gastric juice.

Concerning the free administration of cold water to children suffering with febrile diseases, it would seem supererogatory to say more than a word. The lay mind may balk at furnishing cold water to the child with measles, but the physician who condones such a cruel denial is archaic indeed.

## CHAPTER V

### DISEASES OF THE GASTRO-INTESTINAL TRACT

Of all the diseases of young children with which the physician comes in contact, those referable to the digestive tract will form considerably more than half. In the infants under a year old, and indeed up to the period of complete dentition, these diseases are found more commonly than any others. The very young child is not so susceptible to acute fevers as older children; it is, of course, not prone to contract the various ailments peculiar to adult life, so that we find that dentition, and the various pathologic conditions associated therewith (all of which are exaggerated by diseased states of the digestive tract), and the acute and chronic affections of the stomach and intestines will cause the largest amount of work for the physician during these earlier years of child life. It is of special importance, therefore, that the physician who attempts to treat the various diseases in children shall be familiar with the diseases of their digestive tracts the cause on which these diseases chiefly depend, and the best methods of combating them when they occur.

### THE MOUTH

This orifice plays a very important rôle in the differential diagnosis of the acute exanthemata, on account of the fact that a number of eruptions make their appearance in the mouth two or three days before developing in other parts of the body. In healthy new-born infants the mucous membrane of the mouth is of a pink color, the tongue is usually coated, and there is a very slight secretion of saliva. This continues for the first two or three months of life, toward the end of which time the secretion becomes gradually increased in quantity because of the higher development of the salivary glands. Frequently small flocculi or curds of milk may be seen, and occasionally these must be differentiated from the ulcerative patches of stomatitis or of thrush. Epithelial pearls may also be noted on the gums, and must not be mistaken for pathologic structures. As the epithelium of the mucous membrane is exceedingly delicate, it can easily be injured, and may become a point of entrance for bacteria, thus setting up some form of infective disease.

## DISEASES OF THE MOUTH

## STOMATITIS

**Varieties.**—(1) Simple catarrhal stomatitis; (2) maculo-fibrinous (aphthous) stomatitis; (3) ulcerative stomatitis; (4) gangrenous stomatitis; (5) stomatitis mycosa, or parasitic stomatitis; (6) diphtheric stomatitis; (7) syphilitic stomatitis; (8) mercurial stomatitis.

## 1. Simple Catarrhal Stomatitis

Catarrhal stomatitis consists of a hyperemic condition of the mucous membrane of the mouth, with more or less alteration of its secretion.

**Etiology.**—The causes may be primary or secondary. Among the primary causes are traumatism to the mucous membrane by too vigorous attempts at cleansing the mouth, by the food being too hot, in older children by sharp teeth, or by an irritation or abscess in the gum. In the case of many a child, an excessive amount of candy or cake is almost inevitably followed by such an attack. As secondary causes we have the various gastro-intestinal diseases, teething, eruptive fevers, and quite frequently whooping-cough. Stomatitis is often found in healthy children as well as in those who are sickly. Catarrhal stomatitis is an invariable accompaniment of all other types of inflammation of the oral mucosa.

**Symptoms.**—Preceding the onset of the inflammation in the mouth we may have a slight rise of temperature, some vomiting, constipation—symptoms, in fact, showing the onset of any acute disease. In a short time there will be pain in the mouth or throat, these symptoms occasionally being accompanied by enlargement of the lymphatics under the jaw and in the neck. These symptoms are quickly followed by soreness of the mucous membrane of the mouth, which becomes more and more localized, with anorexia, increased salivation and fetor of the breath.

When the stomatitis is general, the lips share in the inflammation, becoming swollen and tense. A fine papular eruption, caused by the engorgement of the muciparous follicles of the lips, is often seen. The tongue is coated, sometimes slightly swollen.

**Treatment.**—It is often best to give no food for twenty-four or forty-eight hours, simply water or alkaline waters in considerable quantities. When food is given, it should be liquid or semi-liquid and should not be highly seasoned. If the food is given cold, it is often taken better. Weak solutions of sulphate of zinc or salicylate of soda

in a strength of about 1 per cent., or potassium chlorate (2-5 per cent.) are useful local remedies. The agent most generally used, and usually with excellent effect, is nitrate of silver in a strength of about 0.5 to 1.5 per cent.

Applications of alum, made by gently touching the inflamed spot with a single crystal, sometimes give good results. Attention should be directed to the digestion, and the bowels be kept open by laxatives.

## 2. Maculo-fibrinous Stomatitis

**Synonyms.**—VESICULAR STOMATITIS; FOLLICULAR STOMATITIS;  
APHTHOUS SORE MOUTH

Maculo-fibrinous stomatitis consists in a hyperemia of the mucous membrane of the mouth, accompanied by the implantation of small patches of fibrin just beneath the most superficial layers of the mucosa. The overlying mucous membrane then breaks down, leaving superficial ulcers. The term "aphthous," though commonly employed, is somewhat ambiguous, as *apthæ* meant thrush in the old days.

**Etiology.**—This form of stomatitis is most common from the tenth to the thirteenth month of life, although it may occur at any age. The direct causes, are rather obscure although micro-organisms, particularly the staphylococci, are probably the cause. Siegel describes an ovoid bacillus 0.5  $\mu$  in length, which he found in the buccal secretion taken from patients seen during an epidemic in Germany. The disease frequently follows gastro-intestinal diseases, acute fevers, and pneumonia. Preceding the inflammation of the mouth the symptoms of moderate or even high fever, absolute anorexia, increased salivation, vomiting, and constipation—in fact, the same symptoms which may precede any of the acute fevers—may be present. The heat and pain in the mouth increase and there may be some enlargement of the lymphatics. The inflammation of the mouth soon localizes itself into small ulcers of round or oval shape and of yellowish-white color, each ulcer being surrounded by a red areola. As already mentioned, the deposit of fibrin precedes the superficial ulceration. The ulcers may appear simultaneously, or they may come in successive crops. Their most common situation is at the junction of the buccal or labial and the gingival mucous membrane.

**Treatment.**—The treatment consists in opening the bowels with a gentle laxative and regulating the diet. In this form of stomatitis applications of chlorate of potassium, in the strength of from twenty to

twenty-five grains to the ounce, seem to work remarkably well. A favorite prescription of ours is:

R. Potass. chlorat..... ʒi-ʒij  
 Hydrogen dioxid;  
 Glycerin.... āā fl. ʒij  
 Aq. menth. pip..... q. s. ad ft. fl. ʒiij  
 M. et solve.  
 SIG.—As mouth wash every three hours.

The cause of the inflammation, so far as possible, should be removed; the roots of decayed teeth should be taken out, and diseased teeth attended to. The local treatment consists in the application of chlorate of potassium or silver nitrate. Since many of the children affected by this disease are in poor general health, tonics, such as the various preparations of iron and quinin, are indicated. When the mouth is so sore that food cannot be taken in this way, nutritive enemata should be used. The irritation caused by the highly acid secretion of the mouth may be relieved by gently washing out with a solution of borax or bicarbonate of soda. The mouth must be kept clean, but it should be borne in mind that all applications must be applied with great care.

A form of stomatitis only found in the new-born and described under the name of *Bednar's aphthæ* consists in the formation of shallow symmetric ulcers in the mucous membrane over the hamular processes of the maxillary bone. These two ulcers may unite to form a single one crossing the hard palate at these points. This form of aphthæ may be produced by a too violent cleansing of the mouth, although it is occasionally found resulting from the use of badly shaped rubber nipples (Forchheimer). The treatment consists in the application of bland antiseptic washes.

### 3. Ulcerative Stomatitis

"This is a peculiar process, characterized by destruction of tissue, beginning on the gums around the teeth, never extending beyond the mouth, infecting healthy parts of the mouth, and never occurring where there are no teeth" (Forchheimer). The ulcero-membranous affection is dependent upon the fusiform bacillus and the spirillum of Vincent. The ulcers in this affection may also appear upon the tongue, lips or cheeks, and they may accompany an ulcero-membranous angina.

**Etiology.**—Ulcerative stomatitis is rarely found in children under five years of age unless produced artificially by such drugs as mercury.

It usually occurs in children living amid bad hygienic surroundings, particularly when these are associated with poor nourishment. It is occasionally seen following such diseases as scarlet fever or measles, although it is possible for it to occur in any condition in which the health is greatly depreciated. The affection, while not usually serious, is contagious and sometimes runs a subacute and tedious course. A mild form of ulcerative stomatitis of scorbutic origin is occasionally met in infants who have been fed on sterilized milk for a long time.

**Symptoms.**—The disease usually commences with an inflammation of the gums surrounding the molar teeth, or more rarely the incisors, and is most apt to affect those of the lower jaw. The gums become swollen, red, and spongy. In a short time the ulcer extends from the point of origin on the gum to the contiguous mucous membrane of the cheek. These ulcers are usually gray in color, although occasionally they are of a yellowish hue. In a short time sloughing of the tissue follows and the teeth become detached from the gums, the resulting cavity being filled with a mucopurulent secretion. The quantity of saliva is increased, and this secretion, becoming mixed with discharges from the ulcers, produces a peculiar fetid odor in the saliva and breath. In very bad cases the maxillary bones themselves, particularly the inferior one, may be attacked. An eczematous eruption may appear around the lips, caused by the irritation of the saliva. The submaxillary lymphatics become enlarged, but this enlargement rarely tends to suppuration.

In very bad cases the tongue and the entire mucous membrane may become affected, and the part of the latter covering the gum of the lower jaw may be entirely destroyed by ulceration. This is the form of stomatitis which is most commonly associated with scorbutus. If the bases of the ulcers are covered with a membranous deposit, and particularly if the stomatitis accompanies an ulcero-membranous angina (frequently unilateral), Vincent's angina should be thought of and smears should be made.

The prognosis depends on the extent of the disease. When scurvy is the cause, or when any great destruction of bone has occurred, the prognosis is grave.

**Treatment.**—The treatment is both prophylactic and curative. The prevention of the disease is accomplished by improving, so far as possible, the hygienic surroundings of the patient and by the judicious use of good food and tonics. It is well to initiate the curative treatment by swabbing the base of the ulcer with a strong silver solution or with the solid stick of lunar caustic. According to many authorities,

chlorate of potassium is regarded as almost a specific in the disease. It should be given in about 3 per cent. solutions, the effects of the drug being carefully watched. A very good formula recommended by Starr is as follows:

R. Potassium chlorate.....	58 grains
Dilute hydrochloric acid.....	1 fluidram
Syrup.....	1/2 fluidram
Water.....	to make 3 fluidrams.
Sig.—One teaspoonful, diluted in water, for a child three years old.	

When this disease is associated with carious teeth, or if the bone has become involved, the affected structures must be removed. When extreme fetor of the breath occurs, the mouth should be washed out with weak solutions of permanganate of potash or a solution of the hypochlorites. In some cases alcoholic stimulants are indicated. Unquestionably, chlorate of potassium is the most successful remedy in this disease.

#### 4. Gangrenous Stomatitis

**Synonyms.**—CANCER ORIS; NOMA; ORAL GANGRENE;  
WANGENBRAND

Gangrenous stomatitis is a rapidly developing inflammation of the cheek and adjacent tissues, accompanied by gangrene and destruction of the affected parts. It is a rare disease.

**Etiology.**—This form of inflammation of the mouth is rarely seen in children under two years of age. From the second until the twelfth year is the period during which it is most likely to occur. Although the origin is somewhat obscure, it is well nigh certain that it is microbic. The trophic and embolic theories are no longer accepted. Among the organisms that have been found in the affected tissues are the Klebs-Loeffler bacillus and the fusiform bacillus and spirillum of Vincent. It is most common in debilitated children—those long suffering from improper food and bad hygienic surroundings or from the results of some infectious disease. It is particularly common after measles, typhoid fever, and diphtheria, although it may occur after any of the acute exanthemata. The excessive use of mercury is also an occasional cause.

**Pathology.**—Cancer oris presents all the pathologic changes of acute phlegmonous gangrene in any other part of the body. We have here, as in other forms of gangrene, the three zones: In the center is the zone of blackened, destroyed tissue, around the outer margin of which

can be seen the second zone, consisting of connective-tissue cells in a state of active division. The blood-vessels will be found closed by thrombi consisting of various forms of microbic life. The third or outer zone consists of healthy tissue.

**Symptoms.**—During or following convalescence from one of the acute fevers, or in a debilitated child, a small nodule, somewhat hard and sensitive, will appear on the gum or on one of the cheeks. The skin or mucous membrane surrounding it will be either hard and



FIG. 16.—GANGRENOUS STOMATITIS.—(*Dr. Stengel's case at the Children's Hospital.*)

swollen or, as is not infrequently seen when the disease attacks the cheek first, there is simple swelling accompanied by considerable edema of the affected part. Although pain is usually complained of, there is occasionally very little discomfort connected with the progress of this disease. The mucous membrane underlying the external swelling puffs up, forming a vesicle which is filled by an ichorous fluid. This vesicle rapidly changes into a gangrenous ulcer of a blackish or reddish-brown hue. The lymphatics of the neck quickly become infiltrated on the same side as the affected cheek. The skin of the

cheek changes to a bluish color over the point of primary induration. The ulcer rapidly deepens and spreads, first perforating the cheek, then continuing the destruction of tissue until the entire side of the face is destroyed. The disease may involve the whole of the cheek, the neck, and even the eye on one side, but it very rarely becomes bilateral; the bones and teeth of the infected side are entirely laid bare. Gangrene of the mouth is accompanied by great constitutional depression. The temperature is variable: sometimes, considerable fever is found,



FIG. 17.—A FATAL CASE OF NOMA.—Service of Dr. E. E. Graham—(*Philadelphia General Hospital.*)

but as the disease progresses and septicemic symptoms arise we usually find the fever assuming the character found in septicemia elsewhere.

Before death occurs the temperature is occasionally subnormal. The sequelæ of the disease are septic pneumonia, caused by the inspiration of infected material, and diarrhea, also of septic origin. Diphtheria has been observed in a number of cases. As the blood-vessels of the affected part are usually filled with thrombi, perforation of these very rarely causes hemorrhage. Spontaneous recovery from this disease is extremely rare, the mortality being given as from 70 to 90 per cent. of all cases affected.

**Treatment.**—The best treatment of gangrenous stomatitis lies in its

prevention. With this object in view, a child sick with any of the infectious fevers should be placed amid the best hygienic surroundings and its general health kept in as good order as possible by tonics and nourishing food. When the disease has once started, the system should be stimulated by concentrated nourishment, such as meat, milk, eggs, etc. Alcoholic stimulants are particularly indicated here, as in any form of septic infection. As soon as the gangrene appears it should be kept from spreading by a thorough cauterization of the tissues

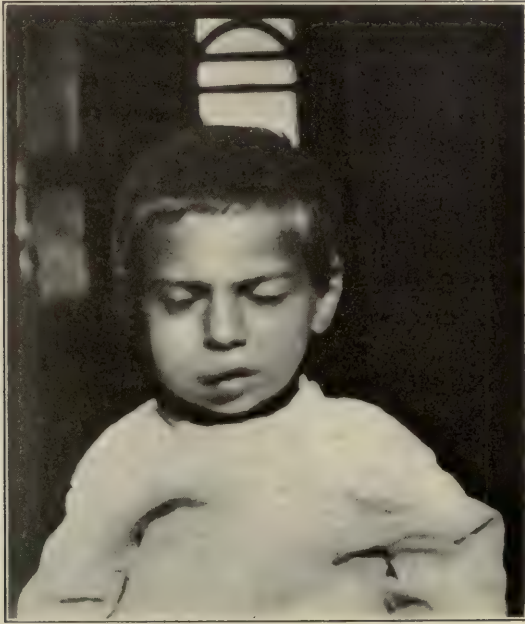


FIG. 18.—BEGINNING NOMA.—The lesions of the cheek and lower lip were gangrenous. From them Dr. Rosenberger recovered the fusiform bacillus and the spirillum of Vincent. The patient recovered under the use of diphtheria antitoxin, and the local use of acid nitrate of mercury.—(*Philadelphia General Hospital.*)

immediately surrounding the ulcer. This can best be done by the use of the Paquelin cautery, the galvanocautery, caustic soda, acid nitrate of mercury, or fuming nitric acid. In order to lessen the horrible stench which arises from the gangrenous tissue the mouth should be swabbed out thoroughly and the ulcer treated with permanganate of potash, carbolic acid, pure peroxid of hydrogen (the latter is very useful), or iodoform and bismuth. In view of the occasional presence of the diphtheria bacillus, and also of the hopeless nature of this disease, diphtheria antitoxin should be very freely used.

### 5. Stomatitis Mycosa or Parasitic Stomatitis

**Synonyms.**—THRUSH; SOOR; MUGUET; SPRUE; MILLET

The disease consists of a yellowish-white deposit of parasitic origin, occurring on the mucous membrane of the mouth.

**Etiology.**—The direct cause of mycotic stomatitis is the *oïdium albicans*. It is probable that this fungus is, in the majority of cases, carried into the mouth by the nipple or the nursing-bottle. Either healthy or unhealthy children may have the disease, but those who have had some slight catarrhal inflammation of the mouth are espe-



FIG. 19.—SYPHILIS SIMULATING NOMA.—There was syphilitic ozena, and a gummatous lesion under the right eye had broken down. There were many other evidences of congenital lues.

cially predisposed to it. Thrush is especially found in poorly nourished infants and in those who are bottle-fed. Any cause producing mechanical injury to the mouth will predispose to the development of thrush fungus. This leads us to lay stress upon the lack of wisdom usually shown in the treatment of the infant's mouth. We heartily agree with the German authorities who disapprove of washing babies' mouths until they acquire teeth. If the food and drink are clean, and the baby has no vomiting, there is no need to cleanse the mouth. Like other forms of stomatitis, it may occur in children recovering from some prolonged illness.

**Symptoms.**—The disease is not often found in healthy infants who are fed upon breast milk, or in those whose bottle feedings consist of proper formulæ furnished in cleanly utensils. It is notably a disease of the institution, of the atrophic baby, and of the baby fed upon a food too rich in carbohydrate. Quite often there are no premonitory symptoms of thrush, the spots themselves being the first indication of the disease. These are of a grayish-white color, of variable size, often “star-like” in form, and rest on the mucous membrane, occasionally, but not always, being elevated above the surface. They appear first on the internal surface of the cheeks and dorsum of the tongue, and then extend backward to the soft palate or forward to the lips. The patches have occasionally been found in the pharynx and esophagus. In very exceptional cases the organisms have entered the blood stream, and have even been found in the brain. An examination of these patches by reflected light shows them to be developed within the epithelium, each being surrounded by a narrow ring of injected blood-vessels. As development progresses the spot is pushed up above the level of the mucous membrane. Occasionally the upper coat of a spot will drop off, leaving quite a deep ulcer. In bad cases a number of these ulcers will become confluent, their covering of fungus forming a sort of membrane. Microscopic examination of scrapings taken from these spots will demonstrate the presence of *saccharomyces* or the *oïdium albicans*.



FIG. 20.—NOMA INVOLVING THE VULVA.

**Treatment.**—The prophylactic treatment of parasitic stomatitis consists in careful attention to the mouth of the child, the avoidance of all forcible attempts at cleanliness, and of abrasions of the mucous membranes. The nursing-bottle and nipple must be kept antiseptically clean. Proper formulæ must be furnished, and sugar in the food reduced to a minimum.

The curative treatment consists in the application to the mucous membrane of solutions of borax or bicarbonate of soda, or boric acid solutions. A very good prescription is the following:

R.	Acid. carbolic.....	gr. ij
	Sodii salicylas,	
	Sodii biboras.....	āā gr. xxx
	Glycerin.....	ʒij
	Aqua rosæ.....	q. s. ad fl. ʒj.
Sig.—For local application.		

## 6. Diphtheric Stomatitis (Croupous Stomatitis)

This rare form of stomatitis may be of primary or secondary origin. When primary, the point of development of the membrane is usually on the lips, extending thence to any part of the mouth. Secondly it may spread from an infected tonsil to the lips, gums, or cheeks. A form of croupous stomatitis may arise from excessive use of irritating drugs used as mouth-washes. In the true form of diphtheric stomatitis the Klebs-Loeffler bacillus will be found.

**Symptoms.**—When the disease is of true diphtheric origin, the symptoms will be those of ordinary diphtheria. Occasionally the symptoms are obscure, and the ulcers may be well developed before they are discovered. The duration of the membrane is usually from three to six days, although it may last longer. Salivation usually forms an accompanying symptom; both the saliva and the breath have a strong fetid odor. As the membrane separates there may be more or less hemorrhage, caused by the exposure of small blood-vessels which have been eroded by the disease. This hemorrhage may sometimes be quite severe.

**Treatment.**—The treatment is the same as that of ordinary diphtheria. When it is possible, the membrane should be carefully removed (never if force is required) and the remaining ulcer washed thoroughly with corrosive sublimate, peroxid of hydrogen, or Loeffler's solution. In the croupous form, resulting from irritation, soothing antiseptic washes should be used. Treatment by the use of injections of anti-toxin should always be resorted to, and the system supported by tonics and good food.

## 7. Syphilitic Stomatitis

The primary infection of syphilis may occur in the mouth, the usual site of the chancre in this case being the lips. The origin of the infection is usually by transmission from a wet-nurse having the disease. The evidences of secondary syphilis of the mouth are not at all uncommon, and may be found on any part of the mucous membrane. The most common forms are those known as syphilitic fissures

or rhagades. These usually occur at the angles of the mouth or upon the upper or the lower lip; they may be single or multiple, and cause considerable pain. These fissures are very slow to heal spontaneously, and even after this has taken place they generally leave a disfiguring scar. Syphilitic papules are also found quite commonly, their seat being the commissure of the mouth and the free borders of the lips. If these split, they cause the rhagades mentioned. In their elevation, position, and in the moisture which covers their surface they resemble condylomata lata. The ordinary mucous patch of syphilis may also be found, either on any part of the mucous membrane lining the mouth or on the tongue.

**Treatment.**—The treatment is that of syphilis generally. The affected parts should be treated by applications of corrosive sublimate, either in the strong solution, applied with a brush, or in a weaker form, to be used as a mouth-wash. The other granulomata may attack the mouth; but such affections are very rare. Tuberculous stomatitis is secondary to lupus, and represents an extension of that disease.

### 8. Mercurial Stomatitis (Ptyalism)

This consists in an inflammation of the mucous membrane of the mouth, attended by a great increase in the quantity of saliva and an alteration in the character of the secretion.

**Etiology.**—The most common cause is the administration of too large a dose of mercury, long-continued use of the drug, or an unnatural susceptibility of the patient.

**Symptoms.**—The first manifestation of ptyalism is an extreme tenderness of the gums, this being particularly felt in biting or in snapping the jaws together. The gums soon become red and swollen, the latter being greatest at the point of insertion of the teeth. There is usually a metallic taste in the mouth, although in children this will not often be complained of, because the patient is too young to associate cause with effect. The secretion of saliva becomes profuse, so that the patient is continually endeavoring to expectorate. The breath is fetid, the tongue swollen, and if the poisoning is severe, the tongue may even protrude from the mouth. Ulcerative stomatitis may follow, and loosening and dropping out of the teeth sometimes occur in severe cases. Complete necrosis of the maxillary bone occasionally follows.

**Treatment.**—The first indication of treatment is to stop the administration of mercurials. In order to increase elimination small doses of potassium iodid may be used, and this may further be aided

by the use of saline laxatives. Frequent bathing and friction of the skin will help in this object. In order to check the hypersecretion of saliva belladonna will be found useful, and if necessary, the drug should be pushed to its physiologic limit. When the pain and distress are great, opium may be used, although great care must be exercised in the administration of this drug, particularly to young children.

Before closing this section on stomatitis, mention must be made of the rare *gonorrheal stomatitis* of the new-born. Practically, it is always contracted from the mother with a gonorrheal vaginitis.

## THE TONGUE

Parenchymatous inflammation of the organ (glossitis) is quite rare in children, but the tongue and mouth often act as indicators for diseases in other parts of the body. Thus we have a blue tongue as a symptom of cyanosis; the pale or colorless tongue seen after severe hemorrhage or in conditions of anemia; the coated tongue found in many diseases of the digestive tract; the red and glazed tongue, with its border of coating, seen in certain fevers. The geographic, or map-like tongue, is also a relatively common find in childhood. Its cause is not always apparent; but many children with geographic tongues are consumers of too much sweet. It should be remembered, however, that no special organisms have been found in the fur of the tongue, and it would be futile to speak of any specific coatings for any one given disease.

## DISEASES OF THE TONGUE

The tongue may be congenitally above or below the normal size, the former condition being known as *macroglossia* and the latter as *microglossia*.

The first is usually found in two forms: (1) The fibrinous, in which the connective tissue of the organ is increased. (2) "A cavernous cystoid degeneration of the interstitial connective tissue, by which the resulting spaces come into connection with the lymph-vessels, constituting a condition closely resembling cavernous angioma, from which it receives its name of lymphangioma cavernosum" (Rotch).

### FIBRINOUS MACROGLOSSIA

**Symptoms.**—The tongue is much enlarged, is of a bluish or violet color and is generally covered with a whitish or grayish coat. Inden-

tations or ulceration of the organ, especially along the edges, may occur from pressure of the teeth. From the size of the tongue respiration and deglutition may be interfered with. The lips, and especially the lower one, become thick, edematous, or ulcerated, and salivation is always present. Macroglossia is most frequently found in deformed subjects or cretins.



FIG. 21.—MACROGLOSSIA.—(*Dr. W. W. Keen's Case.*)

**Treatment.**—The treatment is usually palliative. The tongue should be kept clean with warm, slightly alkaline solutions, or when the organ, by its size, threatens the stoppage of respiration or deglutition, part of it should be removed. Thyroid extract may be tried, and will be found efficacious in the young cretin.

### GLOSSITIS

**Definition.**—An acute inflammation of the parenchyma of the tongue.

**Etiology.**—The disease usually arises from direct injury to the tongue as by teeth, or in the swallowing of irritating substances. The inflammation may occasionally be septic in origin, or it may accompany a mercurial stomatitis.

**Symptoms.**—Pain and swelling of the organ, accompanied by a rise of temperature and a hypersecretion of saliva. Occasionally enlargement of the tongue may interfere with respiration.

**Treatment.**—The treatment is purely symptomatic, depending upon the cause.

## RANULA

Ranula is a cystic tumor of varying size, found on one or the other side of the frenum of the tongue. It may be bilateral, when the frenum of the tongue makes a central depression over the swelling. The tumor is semitranslucent, soft, and over it are seen dilated veins. Its contents consist of a clear, glairy fluid of mucoid character. It is probable that it is due to an obstruction of the ducts of the sublingual mucous glands producing "a congenital or acquired retention-cyst." These tumors generally give rise to little or no pain. They are rather rare in childhood.

**Treatment.**—The contents of the cyst should be emptied, and as recurrence is one of the characteristics of this form of tumor, it is necessary to destroy the lining membrane by the use of caustic. It has been recommended that fifteen minims of a mixture of tincture of iodine and water, of each ten parts, with iodide of potassium one part, will prevent the cyst from filling again. It is probable, however, that a more satisfactory result can be obtained by the application of nitrate of silver to the sac after its contents have been removed.

## DENTITION, NORMAL AND DELAYED

**Physiology of the Development of the Teeth.**—At about the seventh week of intra-uterine life the stratified epithelium of the mucous membrane covering the two maxillæ becomes thickened, forming a ridge. This process passes downward into a recess of the developing embryonic jaw, and is known as the enamel groove. The downward growth, or invagination, of the epithelium forms what is known as the enamel germ, its position being indicated by a slight groove in the mucous membrane of the jaw. Next we find the enamel groove and enamel germ elongating downward, the deeper part declining outward, forming an angle to the upper portion or neck. After this there is an increased development at certain points corresponding to the situation of the future milk-teeth. The common enamel germ now becomes divided at its deeper portion into a number of divisions, each forming what may be called a special enamel germ, which corresponds to what will later be the milk-tooth, in other words, each enamel germ will later form an individual tooth contained in its own dental sac. About this time there grows up from the underlying tissue into each enamel germ a distinct vascular papilla, known as the dental papilla, and upon it the enamel germ adheres. This enamel germ

consists of three layers, or, as it is sometimes described, as two layers of epithelium separated by an interval. While part of the subepithelial tissue is elevated to form the dental papillæ, the part which bounds the embryonic teeth forms the dental sacs, and the embryonic jaw, which at first is merely a groove of bone in which the dental germs lie, now sends up processes, forming divisions or partitions separating the teeth from one another. The papilla is composed of nucleated cells arranged in a meshwork, the outer or peripheral part of which is covered with a layer of special columnar nucleated cells called odontoblasts; these latter form the dentin, while the remainder of the papilla forms the tooth-pulp. As the dentin increases in thickness the papillæ diminish in size, and when the tooth is cut, only a small amount remains as the dental pulp, and in this run the blood-vessels and the branch of the inferior dental nerve, which *enters* the tooth at the inferior extremity of every fang. The enamel consists of three parts: (1) An inner membrane composed of a layer of columnar epithelium in contact with the dentin, called enamel cells; external to this we find one or more layers of small polyhedral nucleated cells; (2) an outer membrane consisting of several layers of epithelium; (3) lastly, a middle membrane, formed of a matrix of non-vascular, gelatinous tissue containing a hyaline interstitial substance. The enamel is formed by the enamel cells of the outer membrane. The development of the teeth progresses steadily from birth during the whole period of infant life. As each tooth, contained in its dental sac and set in its small cavity of bone, develops, elongation takes place, beginning at the fang. In its growth the tooth follows the path of least resistance, which is always toward the mucous membrane, which at the period of birth covers it. Finally, from pressure against the mucous membrane, atrophy or absorption of the latter takes place and the tooth appears above the level of the mucous membrane.

As the child advances in years the temporary or milk-teeth are gradually replaced by the permanent teeth, which push their way up from beneath the former, absorbing in their growth the whole of the fang of every member of the first set until little is left except the crown, which finally comes away.

The age at which the first tooth appears varies considerably, this depending upon many causes. As a general rule, in healthy children the first tooth appears about the sixth to the eighth month. The eruption of the teeth begins later in children affected by rachitis, syphilis, or tuberculosis, or in those who may be classified under a general head as being "feeble children." Dentition may be delayed for months, or even

years, in Cretins and Mongolian imbeciles. The lower central incisors usually appear first, and from this time dentition may be divided into five periods, between each of which is an interval of varying length, sometimes known as interdental intervals. The *first period* occupies the time when the two lower incisors are cut. In the *second period* the four upper incisors make their appearance, these being followed very often by an interval of several weeks. The *third period* is that in which the lower lateral incisors and the anterior molars of the upper and lower jaw are cut; this lasts from the twelfth to the fourteenth month, and is usually followed by quite a long interval of rest. The *fourth period* begins at about the eighteenth or twentieth month, and it is at about

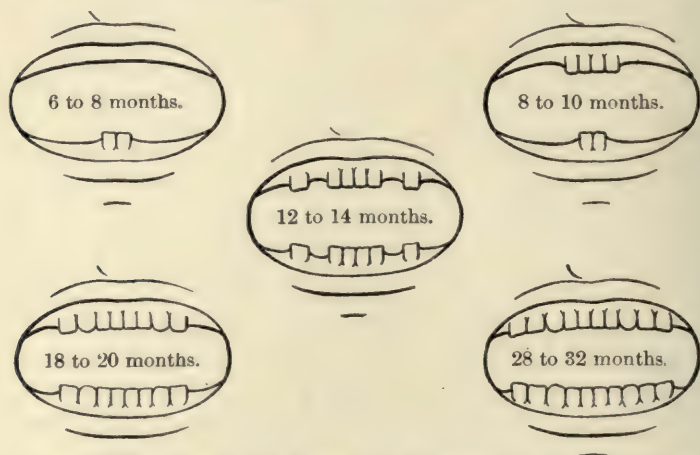


FIG. 22.—DIAGRAM SHOWING THE ORDER OF THE ERUPTION OF THE MILK-TEETH.

this time that the canines appear. The *fifth period* occurs at two and a half years of age, when the posterior molars are cut. The entire "milk-set" is composed of twenty teeth, ten in each jaw, arranged as follows:

DENTAL PERIODS	AGE	GROUP OF TEETH
I.....	6 to 8 months,	Two middle lower incisors.
II.....	8 to 10 months,	Four upper incisors.
III.....	12 to 14 months,	Two lower lateral incisors and four first molars.
IV.....	18 to 20 months,	Four canines.
V.....	28 to 32 months,	Four second molars.

At birth the jaw contains the entire milk-set, the crowns of which are calcified. Besides these there is one member of the second set—the six-years-old molar—the calcification of which begins during uterine life, at about the sixth month. The permanent incisors begin

to calcify during the first month of life; the canines in the first or second year. Calcification of the crown of the second molar is completed about the fourth year, and of the third permanent molar, or wisdom-tooth, at from seventeen to twenty-five years. Thirty-two teeth in all comprise the second or permanent set, and during their development the jaws increase in length to provide for the greater number of teeth. The second dentition cannot be divided into so clearly marked periods as the first, but the ages at which the teeth make their appearance are, generally speaking, as follows (it must be remembered, however, that as many causes may delay the eruption of the first teeth, so may many retard the second set). They are not necessarily the same causes, however.

YEARS	GROUPS
Six.....	Four first molars.
Seven.....	Four middle incisors.
Eight. ....	Four lateral incisors.
Nine.....	Four first bicuspid.
Ten.....	Four second bicuspid.
Eleven.....	Four canines.
Twelve .....	Four second molars.
Seventeen to twenty-five.....	Four third molars (wisdom-teeth).

Dentition being a physiologic process, there should be no symptoms of a pathologic nature dependent upon it. It is true that various disorders, principally referable to the nervous system, the respiration, the skin, the digestive system, and organs of special sense, have been associated with it, but if the child at the time of dentition is in a thoroughly healthy condition, there should be no bad symptoms other than some irritability of temper, restlessness, and possibly a slight disturbance of the alimentary tract. Even the latter is by no means a necessary consequence, though usually there is lessened appetite. The gastro-intestinal tract is more predisposed to fermentative diarrheas during dentition, but these are not primarily caused by teething, but by infection from without, the intestinal tract being in a more irritable condition at this time, and therefore less resistant to external sources of infection.

One of the complications of dentition most dreaded by mothers are convulsions. Leaving out of the question those children with neurotic histories or those whose constitutions have been weakened by rachitis, inherited syphilis, or tuberculosis, we cannot now recall a single case in which teething has primarily caused convulsions. The eclamptic seizure presupposes an underlying, as well as an exciting cause. Us-

ally one finds behind that paroxysm, rickets or neurotic inheritance. In the majority of patients who have come under our notice with convulsions during dentition the cause could almost invariably be traced to an attack of acute indigestion in a child whose digestive apparatus was in a hypersensitive state at this particular time; the primary cause,

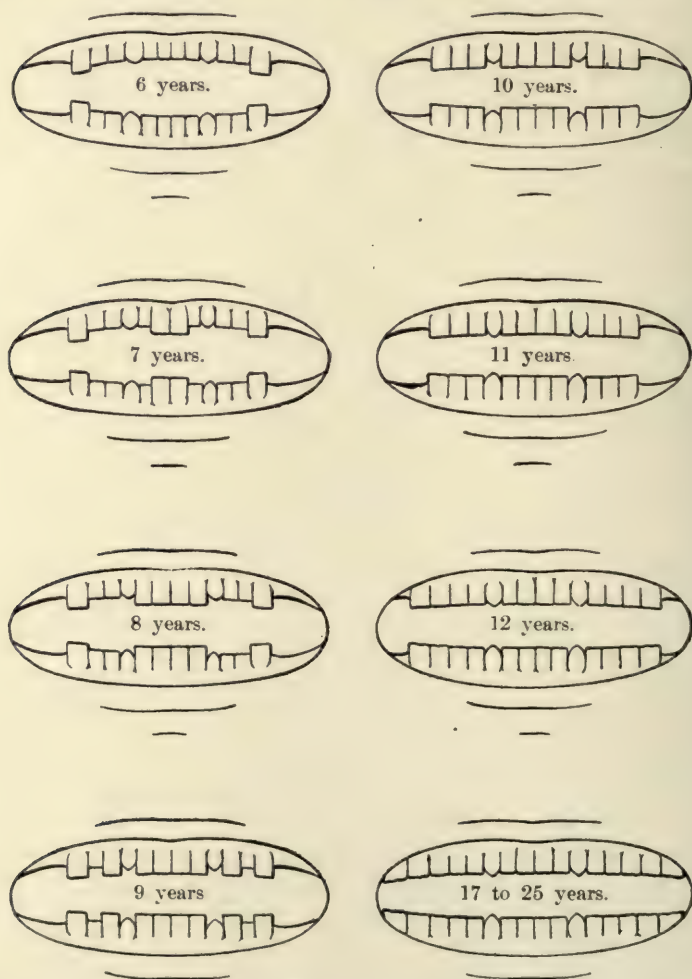


FIG. 23.—DIAGRAM SHOWING THE ORDER OF ERUPTION OF THE PERMANENT TEETH  
—(From Rotch.)

however, has been a badly regulated diet, and not the cutting of the tooth. Bronchitis and bronchopneumonia have also been ascribed to dentition. It is true that a slight irritation of the faucial or nasal mucous membrane may, in a sensitive child, be produced by sympa-

thetic irritation; but here again we must say that we have never seen a case of either of the before-mentioned diseases arise from dentition. A slight erythematous rash, formerly known as tooth-rash and still associated with dentition in the minds of parents, sometimes covers the body at various intervals during this period, but as children affected by it have always at the same time had gastro-intestinal disorders, we cannot believe that dentition was responsible for the cutaneous affection. Slight facial eczema may occur with the eruption of every tooth. In children affected by struma or in rachitic babies it is not uncommon to find enlargement of the glands of the neck or some irritation of the ears.

Ordinarily a healthy child will evince no symptoms severe enough to demand any special treatment. When, however, an infant is feverish, restless, and irritable, it is proper that the physician endeavor to quiet this condition by suitable medicinal means. Small repeated doses of bromids with camphor water, or, what we have found more useful, small doses of antipyrin, will generally bring about this result. A prescription which we have frequently employed is as follows:

R. Sodii bromid.....	gr. ij
Antipyrin.....	gr. ss-j
Glycerin.....	ʒiij
Spirit. menthæ piperitæ.....	gtt. ss-j
Aqua camph. (or soda-mint mixture).....	ʒj.

Sig.—To be repeated every two or three hours until the child is quieted.

During the period of dentition the child's diet should receive the strictest care, as it is at this time that the most virulent forms of intestinal diseases frequently make their appearance. The "second summer," which is looked upon with so much dread by mothers, is frequently coincident with the period of active dentition, and it must be borne in mind that at this time the nervous system is usually in a more or less hypersensitive condition, and therefore the digestive system, sharing in this irritability, is less able to combat the attacks made on it by external causes of infection. The proverbial fear of the second summer is readily explained by the fact that the child is then receiving artificial food, much of which (cow's milk) may teem with bacteria. Artificially fed babies are in more danger during the first summer, however, than during the second. Children whose systems are enfeebled from constitutional diseases should be appropriately treated, and rachitic or strumous patients, who are apt to be especially depressed at this time and are always slow in cutting their teeth, should be treated

by the administration of oils and such remedies as phosphorus and the salts of calcium and general tonics. When a tooth is slow in coming through the gum and its eruption is accompanied by a decided and continued rise of temperature or severe nervous symptoms, lancing the gum is often beneficial. The same treatment should be adopted in acute illness when difficult dentition accompanies it. Unless the physician has a clear idea of the exact position of the tooth which is causing the trouble, gum-lancing is of very little use and simply produces an open wound, which always offers an entrance-point for infective germs. The custom of indiscriminate gum-lancing is to be condemned.

## DISEASES OF THE ESOPHAGUS

### ESOPHAGITIS

Inflammations of the esophagus are of rare occurrence in children. It is stated that inflammations of the mouth, although very common, extend but rarely into the esophagus.

### Acute Esophagitis

**Etiology.**—Inflammation of the esophagus may be caused by lacerations produced by swallowing a foreign body or by corrosive poisons. Either of these causes may involve the mucous membrane or may extend into the muscular coat. The latter is, however, of rare occurrence. When a foreign body has been swallowed, the early symptoms are pain or swelling and a general discomfort in the region. When strong acids or caustic alkalies have been taken into the mouth, the mucous membrane of the latter will be inflamed and ulcerated, the character of both varying somewhat with the poison taken. There will be a burning pain in all the parts affected, great thirst, and a spasm of the esophagus following attempts at swallowing. Deglutition is almost impossible. The period of acute inflammation will last several days, and during this time there is often great danger of suffocation from edema of the glottis. Later, symptoms of stricture are very common, these usually occurring in from three to six months after the injury. One of us exhibited before the Philadelphia Pediatric Society, a child four years of age, who had swallowed lye several months before. She was operated upon and fed through a gastrotomy tube for two years. She eventually perished of tuberculosis of the bronchial glands.

**Treatment.**—The indications for treatment are to remove the foreign body, and when the inflammation is produced by poisons, to neutralize the latter as quickly as possible by the use of oils, demulcent drinks, and ice. For the pain, opium is indicated. The treatment of stricture of the esophagus is surgical.

Post esophageal abscess is occasionally seen, but not nearly so commonly as the postpharyngeal affection. It is usually an accompaniment of Pott's disease.

The writers cannot lay too much stress upon the importance of digital examinations of the nasopharynx and the upper esophagus in obscure cases. One of us so detected a jackstone in the esophagus a very short time ago.

## DISEASES OF THE STOMACH

### GASTRITIS

**Synonyms.**—ACUTE GASTRIC CATARRH; ACUTE DYSPEPSIA; GASTRIC FEVER; GASTRO-ADENITIS

**Varieties.**—Acute gastritis; chronic gastritis; ulcerative gastritis; gastromalacia.

**Acute gastritis** is an acute inflammation of the glandular tissue of the stomach, interfering with the digestive functions, and generally due to the presence of irritating ingesta (Blackader).

**Etiology.**—By far the most frequent cause is food improper in kind, quantity, preparation, or time of use. Although acute gastritis rarely occurs in young children fed on breast milk, yet it is occasionally seen. In most cases the attack can be traced to some morsel of coarse "table food," candy, or cakes. The substitution of artificial feeding in nurslings, especially when the food is improperly prepared, is another frequent and powerful cause. Unsterilized water often produces acute dyspepsia. An attack of acute gastritis sometimes precedes the eruption of a tooth, although in many instances it is hard to prove whether the attack is due primarily to disordered dentition or to some error in diet during this epoch when the whole digestive tract is in a more or less irritable state.

In older children large quantities of food taken at one time, late meals, highly spiced articles, too great a variety of food, and very rapid eating are the most common causes. Children born with an enfeebled nervous system and those reared on proprietary foods are particularly liable to gastritis. Sudden chilling of the skin has been shown to be an occasional cause.

**Symptoms.**—At the onset of an attack of gastritis a child who has previously been reasonably healthy loses interest in its play and surroundings. If asleep, it may awake crying and complaining of pain, usually referred to the abdomen. In young children the thighs are then flexed on the abdomen and the legs on the thighs; the arms also are flexed. The crying is sharp, shrill, and continuous, and around the mouth and chin there is generally a pale-blue line. In the febrile type the temperature may rise to  $102^{\circ}$  to  $103^{\circ}$  F. ( $38.9^{\circ}$  to  $39.4^{\circ}$  C.). The pulse and respiration are both accelerated. Vomiting is an almost constant symptom, accompanied by nausea and full unloading or retching; this vomiting must never be mistaken for the simple regurgitation of milk which occurs when the infant has taken more than the stomach can hold.

Upon examination of the vomited matter hydrochloric acid will be found to be deficient. The emesis may continue for a considerable time after the contents of the stomach have been evacuated. The tongue is usually covered with a white or brownish-white coat; this coating is particularly heavy at the base of the tongue, while the tip and edges of the latter are a bright red. There is complete loss of appetite, and when the intestines become involved (as they nearly always are to some extent), there will be diarrhea, with the expulsion, first, of the normal contents of the bowels, and, later, large quantities of mucus. Occasionally the attack is ushered in by convulsions, which are sometimes quite severe, and one of the authors remembers a child five years of age who, forty-eight hours after having eaten a large variety of cakes and candy, was taken with violent convulsions of an eclamptic character. Unconsciousness was to all appearances profound; the temperature and pulse were both considerably above normal, and the type of spasm was first tonic, followed by a long-continued series of clonic movements. The symptoms rapidly disappeared after thorough evacuation of the stomach and bowels, wrapping the child in a hot wet-pack, and stimulation of the rapidly failing heart by hypodermic injections of strychnin and atropin.

Other symptoms of acute gastritis which are noticed particularly in older children are: Tenderness in the epigastrium, with a moderate distention of the abdomen, often causing pain or uneasiness on the slightest touch; headache, which may be general or confined to the frontal or occipital regions; occasionally pharyngitis may appear. Phenomena simulating profound nervous disturbance may occasionally be met; thus, Seibert has reported a number of cases in which the symptoms of gastritis closely resembled those of cerebral meningitis, and instances in which

aphasia and hemiplegia followed the eating of a large amount of indigestible food are reported by Henoch, Fraenkel, and others. When the inflammation extends as far as the duodenum, an attack of catarrhal jaundice may follow within a day or two. If the attack is not very severe, the symptoms will subside rapidly in from one to three days. When severe or when the cause is not quickly removed, the stomach may remain inflamed for several days, the gastric irritation being accompanied by fever and evidences of rather severe exhaustion.

**Pathology.**—The mucous membrane of the stomach appears swollen and reddened. In severe attacks erosions and even slight hemorrhages may be found. The tissue beneath the mucous membrane—the submucosa—will be found edematous; when seen through a microscope, the interstitial tissue is infiltrated with leukocytes, and the differentiation between the parietal and principal cells cannot be made out. All the cells appear cloudy and granular and partially separated from the membrana propria of the gland. There is a great abundance of the mucous cells in the pyloric region, and this increase extends deeply into the ducts of the gland.

**Diagnosis.**—This must be based upon the history and the symptomatology as outlined. It is always necessary, however, to exclude vomiting dependent upon other causes, such as the following: The onset of an acute infectious disease, particularly scarlet fever, pneumonia, tonsillitis, and meningitis; the ingestion of a mineral poison (phosphorous poisoning from match heads, etc.), appendicitis, intussusception, strangulated hernia or some other form of intestinal obstruction; cyclic vomiting, congenital malformations of the intestinal tract or bile ducts, nephritis, etc.

**Prognosis.**—The prognosis of acute gastritis is good, except when the child has for a long time been badly nourished. It is less favorable in bottle-fed babies and in older children when the attack is complicated by severe convulsions. If nephritis is coexistent, or when the attack occurs at the end of one of the continued fevers, the outlook is not so good.

**Treatment.**—In an attack of acute indigestion the first indication is to remove the irritating material as quickly as possible. With this object in view the vomiting should not be checked unless it produces severe exhaustion. Indeed, very frequently it is well to aid the expulsion of undigested food by the administration of emetics, such as weak mustard-water or ipecac. Ipecac is best given in the form of syrup or wine, in teaspoonful doses, repeated until vomiting occurs. In some cases it will be necessary to wash out the stomach

by means of a stomach-tube. The bowels should be evacuated thoroughly by means of some mild purgative, and probably for this purpose there is no drug so useful as calomel. It has been our experience that this agent can be given with much better effect in small doses repeated at short intervals until the desired action is obtained. A useful formula is the following:

R̄.	Hydrarg. chlorid. mite.....	gr. iiij
	Pulv. ipecac.....	gr. vj
	Sodii bicarb.....	ʒ ij.
	M. et div. chart. No. xxx.	

SIG.—One powder every hour until the stools change to normal color.

It is often good practice, after having used these powders for twelve hours, to administer a dram or two of castor oil. Young children often take this agent remarkably well; when, however, the child objects to taking it, the oil may be sandwiched between two thin layers of some tart jelly, or it may be given with a few drops of whisky, or floated on iced water. In older children any of the formulas given below may be found useful:

R̄.	Hydrarg. chlorid. mite.....	gr. ii-v
	Sodii bicarb.....	gr. xii-xxx.
	M. et div. chart. No. viii.	

SIG.—One powder every two hours until the bowels are freely evacuated. For a child from two to four years old.

R̄.	Sodii et potassii tartratis .....	gr. xx-xl
	Sodii bicarb.....	gr. iii-vj.
	M. et div. chart. No. vi.	

SIG.—One powder to be given in a wineglassful of hot water every hour or two until the bowels are freely evacuated.

R̄.	Hydrarg. cum creta.....	gr. vi-viiij
	Sodii bicarb.....	gr. viii-x
	Pulv. rhei comp.....	gr. xij.
	M. et div. chart. No. iv.	

SIG.—One to be given every two hours until the bowels are emptied.

The intestinal antiseptics, such as salol, beta-naphthol, naphthalene, and phenolphthalein, given in doses suitable for the age of the child, are very useful. Beta-naphthol bismuth, in doses of from one to five grains, according to the age of the child, and repeated every three or four hours, has given good results. When the vomiting is persistent, minute doses of calomel mixed with bicarbonate of soda or triturated with sugar of milk are very beneficial. Small doses of 1/10 of a grain of calomel combined with from 1/500 to 1/200 of a grain of arsenite of copper have, in our experience, worked admirably

in checking several cases of severe vomiting. When the vomiting continues for some time after the stomach is empty or immediately follows the taking of food, from one to five drops of tincture of nux vomica, given just before feeding, will often stop further trouble. Small doses of sulphate of magnesium have been recommended by Stuart Patterson in the treatment of this condition.

Injections into the bowel of a sterilized .6 per cent. solution of chlorid of sodium are particularly useful, and can be relied on for most cases. Of the greatest importance in acute gastritis is the regulation of the diet; during the attack the stomach should be allowed absolute rest, no food being given. In bottle-fed babies, particularly during the summer months, milk and all articles containing milk should be omitted. For the first twenty-four to thirty-six hours the infant should receive nothing but cold sterilized water in quantities of from half an ounce to an ounce every three or four hours, slowly administered. This water may contain about twenty or thirty drops of good whisky or brandy. When the vomiting and diarrhea have ceased, the child may receive small quantities of some good beef-extract or thin, strained broth. Barley water or rice water may also be allowed in small portions. When meat extracts cannot be borne by the stomach, albumin water, made by mixing the whites of two fresh eggs in a glass of water and having in it a little salt, will sometimes do very well, and occasionally koumiss is well retained by older children. For the latter the diet should consist of thin broths, and when the intestines are not involved, small quantities of starchy foods may be allowed. When fever is present, the child should be occasionally sponged with cool water containing alcohol or ammonia. The customary diet of the child should be resumed by degrees. Many children recover much faster if they are sent to the country, or particularly to the seashore. Frequently even the change of a visit to the house of some relative near by will prove beneficial.

### CHRONIC GASTRITIS

**Synonyms.**—CHRONIC GLANDULAR GASTRITIS; CHRONIC VOMITING

This disease consists in a chronic inflammation of the mucous membrane lining the stomach, attended by hyperemia and thickening of the mucosa, giving rise to a decrease both in the quantity and quality of the true glandular secretion of the stomach, the gastric juice. As a result of the chronic catarrh large quantities of adherent mucus of a strongly alkaline reaction are formed. This results in an

enfeeblement of the digestive powers of the stomach, which, in turn, frequently causes retention of food and consequent fermentation.

**Varieties.**—Chronic infantile gastric catarrh; chronic gastro-enteritis.

**Etiology.**—A very common cause is the continuation of an attack of acute gastric catarrh, the treatment of which has been neglected. The too hasty mastication of food, eating at too frequent intervals, a diet unsuitable for the child, such as very rich, improperly cooked, or highly seasoned foods; the continued use of candy, cakes, fried foods, or hot breads; general bad hygiene, uncleanness, and the constant use of starchy foods or those containing too large an amount of sugar are frequent causes of chronic gastritis in infants. As predisposing causes we have syphilis, rachitis, scrofula, and a low degree of inherent vitality. Diseases of the heart, lungs, liver, and kidneys may also act as predisposing causes.

The repeated swallowing of infected discharges from ulcerations in the mouth, throat, or nose, or the mucus from chronic nasopharyngeal catarrh, and carious teeth may also act as causes.

**Pathology.**—The pathologic changes in chronic gastritis are of the same nature as those in the acute form. The mucous membrane becomes thicker as the disease progresses, its color turns grayish, with deeply injected areas. The whole membrane is covered with patches of dense, sticky mucus. Throughout the whole mucosa, but particularly in the region of the pylorus, we find small papillary projections caused by the hypertrophy of the mucous membrane. This condition is sometimes known as *etat mamellone*, and may, in very bad cases, advance to such an extent as to produce absolute polypoid growth. The gland cells may be destroyed in patches, rendering the differentiation between the principal and parietal cells impossible. If the disease progresses, an infiltration of small cells takes place, with loosening and separation of the superficial layer of the epithelium. According to Ewald, there is a mucoïd transformation of the cells of the tubules, which may extend to the base of the gland. In very advanced cases there is a progressive fatty degeneration of the cells, finally ending in an acute atrophy of the mucous membrane.

**Symptoms.**—The attack is usually a simple continuation of the symptoms of acute gastritis. The vomiting at first is of the contents of the stomach, then of sour, bile-stained mucus, and, finally, when this symptom continues, there will be simply an ejection of clear, watery fluid, sour smelling and tasting, frequently mixed with fragments of food. Vomiting is increased after the taking of food, and in

young infants particularly after the ingestion of farinaceous foods. Vague feelings of distress and pain are felt, referred to the abdomen, the infant generally lying with its legs drawn up. The abdomen itself is usually distended and distinctly tender to the touch. As a general rule the bowels are constipated, although there may be occasional short attacks of diarrhea, during which considerable quantities of mucus are passed. Eructations of gas are common, particularly after feeding. The tongue is coated, this coating being greatest at the back and center of the organ. The papillæ are enlarged and the edges and tip are of a bright glazed red.

The skin in children suffering from chronic gastritis is dry and scurfy; this symptom is particularly noticeable on the surface of the scalp. Various irregular forms of skin eruptions may appear. Several of the forms of stomatitis are not rare, the parasitic variety being that most commonly met. The general condition of these patients is poor, and their appearance is that of chronic ill health. They are thin, pale, with sunken eyes, depressed fontanels, and, when the disease is prolonged, show the characteristic angular face of marasmus. Under the eyes and around the mouth will be seen the blue line so common in chronic affections of the digestive organs. The appetite is generally poor, although it is not at all uncommon for a child who has refused food when offered at the proper times to ask eagerly for all sorts of odd articles of diet between meals and at night. These children sleep poorly; at night they are often disturbed by frightful dreams, from which they start wildly and cry out; incontinence of urine is very common. Frontal headache is a frequent symptom, and choreic movements not seldom occur. Otitis media and abscess of the middle ear may occur, though not unless there are accompanying adenoids. Cases of apparent loss of consciousness and symptoms resembling petit mal due to chronic gastritis have been reported. Attacks of irregular heart action are very frequently met, but this symptom appears chiefly in older children. These children seem to be continually deficient in bodily heat, have cold feet and hands, which unless they are surrounded by external warmth, feel to them icy cold. From various reflex irritations we may have a dry hacking or, as is occasionally heard, a loud ringing, cough, somewhat paroxysmal in character and increased at night or after taking some article of indigestible food. Various intestinal irritations simulating worms may also be present. Occasionally a slight rise of temperature is noticed in the afternoons.

**Diagnosis.**—The diagnosis is founded on the long continuance of

the disease, the chronically disturbed digestion, bad nutrition, and the exclusion of organic diseases of the heart, lungs, and kidneys. The disease with which it is most likely to be confounded is tuberculosis, especially when the latter has reached an advanced stage; but in tuberculosis we have involvement of the lungs, and a greater and more constant rise of temperature. The cutaneous or per-cutaneous tuberculin reaction may prove useful. The finding of the bacillus tuberculosis in any of the discharges would settle the diagnosis.

From syphilis it can be differentiated by the fact that in specific disease we have the characteristic eruptions and many other symptoms of this affection. In any doubtful case the application of antisiphilitic remedies or the Wassermann test will settle the question of the diagnosis. Typhoid fever can be differentiated by the character of the stools in the latter disease, the fact that young infants are not very susceptible to typhoid fever, the characteristic temperature range, and the greater severity of the attack. Widal's blood test will conclusively prove the diagnosis of typhoid fever.

**Prognosis.**—Under proper diet, care, and hygienic surroundings the outlook for children affected with chronic gastritis is fairly good. The prognosis is rather worse during the teething period, particularly if this occurs during the summer months. It should not be forgotten that while chronic inflammation of the stomach is not often fatal itself, yet it so lowers the vitality of the child as to render it an easy prey to other diseases.

**Treatment.**—The first indication of treatment is carefully to regulate the diet, feeding the child at as regular intervals as possible, and far enough apart to give the stomach a period of absolute rest between them. Of scarcely less importance are the general hygienic surroundings of the child, its bath, clothing, and general mode of life. In selecting a diet for these cases we must pick out one which will adapt itself to the portion of the digestive tract which is the healthiest. Infants who have been fed on farinaceous foods or condensed milk, those who have nursed from the breast of an unhealthy mother, or who have been given the breast at irregular intervals, so that their stomachs are kept in a continually overloaded condition, should have their diet strictly regulated. Infants who have been fed on artificial foods should be placed on a diet of modified milk, the formula of which may have to be changed many times; or, if that disagrees, they should be fed on small and carefully regulated quantities of animal broths or extracts or predigested milk. Breast-fed babies should be nursed at exact intervals of from two and one-half to three hours,

and between these periods no food whatever should be given. In older children a carefully prepared bill of fare should be directed by the physician each day. This diet-list (best written down) should carefully avoid all rich or highly seasoned foods or a great variety, although a certain amount of change in diet is as important to a child as it is to an adult. The particular kind of food must be selected by the physician for each individual case, for it is impossible to give definite rules as to the diet proper for these children. It is of importance, however, that the heaviest meal should be given in the middle of the day, and that a very light supper, consisting of crackers and warm milk or a small quantity of thin strained broth, should be eaten at night. The last meal should be taken not less than an hour before retiring. For more careful directions as to the feeding of these children the reader is referred to the chapter on the preparation of foods. These patients should be kept as much as possible in the open air; although they do well in the country, yet the seashore is decidedly the best place for them. It is of considerable importance in selecting a place for their convalescence that one be chosen where the drainage is at least fairly good. On account of the hyperesthetic nervous condition which almost always accompanies chronic digestive diseases of all kinds, these patients should be kept as much as possible from frights and nervous shocks. They should not be put to school too early, or subjected to the long-continued hours of study which children usually have to undergo. The daily bath is a matter of considerable importance. Each night and morning the patient should be sponged off with water at a temperature of about 86° F. (30° C.). Sponge-baths of sea-water or water containing rock-salt are of great value. Wiederhofer recommends that as soon as a child is out of bed in the morning it should receive a good rubbing with a rough towel. It should then stand in the bath, which contains warm water three or four inches in depth, and be sponged down as quickly as possible with cool salt water, and a half a gallon of the same be emptied over the chest and shoulders. The child should again be rubbed dry until the skin is well reddened. Long hours of sleep are of the utmost importance to these patients. Massage of the abdomen and the application of faradic electricity are extremely useful adjuncts in the treatment.

The indications for medicinal treatment are, first, to rid, as far as possible, the mucous membrane of the adherent mucus which covers it, and then to stimulate it to a secretion of healthy gastric juice. In order to accomplish the former the practice of washing out the stomach by means of a stomach-tube and funnel is of the greatest use, and

should be repeated three or four times a week. When this cannot be borne, as in cases in which it excites persistent vomiting or when organic, cardiac, or pulmonary disease exists, lavage may be substituted by warm alkaline drinks or small quantities of alkaline mineral water. Calomel in these cases is a remedy of great usefulness; small doses may be given in combination with bicarbonate of soda. Potassium-tartrate of soda, or phosphate of soda, given three or four times a day has peculiar tonic value. When diarrhea occurs, calomel with salol or beta-naphthol bismuth may be used. In some cases the sulphate of magnesia is highly recommended to relieve the constipation of this disease, and an occasional single dose of castor oil acts happily to cleanse the whole intestine. Hydrochloric and nitrohydrochloric acid in doses suitable to the age of the child are very useful agents. The various bitter tonics, such as nux vomica, gentian, or quassia, are valuable in these cases.

### CYCLIC VOMITING

Cyclic vomiting is an affection of unknown origin occurring most often in children of gouty or neurotic tendencies, and characterized by periodic and recurring attacks of severe vomiting. The attack does not necessarily follow acute indigestion, but is accompanied by deficient excretion of uric acid, by the presence of acetone, diacetic acid and oxybutyric acid in the urine (Edsall), the presence of an acetone odor on the breath, and by severe prostration, from which the patient rapidly recovers.

**Etiology.**—The direct exciting cause has not been discovered, but it seems to be associated with a general derangement of nutrition and assimilation. It usually occurs in families of the better classes, and sometimes in several children of the same family. As mentioned, these children are often neurotic. It usually begins somewhere between the second and the fourth years of life, though it may first appear in infancy. It affects both sexes with equal frequency. Edsall considers it an acid intoxication. Exhaustion, fatigue, overstudy, or overwork seem to act as predisposing causes.

**Symptoms.**—The attacks, which recur at intervals varying from a week to several months (usually three or four a year), are usually preceded by a prodromal stage lasting from twelve to twenty-four hours. During this period the child will be languid and dull, with loss of appetite, occasionally constipation, and a sense of general discomfort in the epigastrium. The temperature is not usually elevated, though slight

risers are more common during the prodromal period than during the attack. The excretion of uric acid is considerably under normal. Acetone is commonly present in the urine and the odor of acetone is noticeable upon the breath. At the end of the prodromal stage the child is suddenly seized with vomiting, at first after taking food or drink, but soon the vomiting becomes almost constant, or in some cases there may be an interval of a half hour or so; it is accompanied by great retching and distress. The vomited matter consists of frothy mucus and serum, which may occasionally be blood-streaked. The reaction is highly acid. After the beginning of the vomiting the temperature is rarely elevated, or if so there is only a subfebrile rise. From the severe vomiting the child becomes exhausted to a degree which may excite alarm; the pulse is rapid, weak, and sometimes irregular. One who has never seen a case of this disease cannot begin to appreciate the apparent seriousness of the clinical picture it presents. After twenty-four hours of vomiting, or indeed, in less time, the little sufferer is in a stuporous or semistupid condition. The fontanelle, if still open, is depressed. A hollow appearance is presented around the eyes, and dark circles exist on the lower lids. The nose presents a pinched appearance. The tongue is dry, and heavily coated, often with a brownish fur. The odor of acetone is usually present in the breath. The breathing is shallow and slow, sometimes sighing in character. The heart sounds are usually weak. The abdomen is flat, soft, and frequently retracted. The liver may be slightly enlarged, however. The amount of urine is much diminished. Important urinary finds have been mentioned; but in addition to these, one may detect albumin, hyaline casts and indican. The bowels remain obstinately constipated, even enemata moving them but sparingly. The attack is usually severe for twenty-four hours, and persists with less intense and frequent vomiting for three or four days. Some cases last a week. As the attack draws to a close the vomiting becomes less frequent and severe and the patient gradually recovers—indeed, at the end of the period the child is often better than before the attack began.

**Diagnosis.**—Cyclic vomiting may be differentiated from acute indigestion by the fact that in cyclic vomiting the attacks are not brought on by indigestible food, and by the persistence of the vomiting. From gastritis it can be distinguished by its short duration and self-limited course. The diagnosis between cyclic vomiting and that of organic disease of the kidneys must be founded on a careful study of the urine. Meningitis should be differentiated by the fact that the

general history of the two diseases differs. In cyclic vomiting there is a history of repeated attacks occurring over a considerable period of time.

**Prognosis.**—The outlook for life is usually good, though Griffith and others have recorded fatalities. Hygienic and dietetic measures may do much for these children, but the parents should be warned that recurrences may take place for years.

**Treatment.**—When the patient is seen during the preliminary stage, free purgation by calomel may have a good effect. The alkaline treatment of Edsall may accomplish much during the attack, though in other cases it may disappoint. It is our custom to give carbonated waters, ice-cold and in very small amounts. It is true that any liquid may incite vomiting, but surely it is easier to vomit when the stomach contains something. More than that, the vomiting is apparently nature's effort to get rid of some noxious element. Brandy in small doses we usually add to the carbonated drinks. Brandy is also added to the normal salt enteroclyses. The latter should be given at least twice daily, and as much fluid as possible should be retained in the bowel. Hypodermoclyses should also be employed when the case is a desperate one. Koplik has found codeine of value in this disease. The chief indication seems to be to prevent recurrence of the attacks, and on general principles as much exercise in the open air as possible should be secured. Too long hours of school and overstudy generally should be forbidden. Children's parties or indeed anything unduly exciting in the child-life should be prohibited. The diet should be carefully regulated, allowing a minimum of meats and a considerable quantity of fruits, either fresh or stewed, and simple farinaceous foods. Edsall's alkaline treatment is again worthy of trial.

#### PYLORIC HYPERTROPHY WITH STENOSIS

Congenital hypertrophy of the pyloric end of the stomach with stenosis of the pylorus is a somewhat rare condition, but one of us has observed seven cases.

**Etiology.**—The condition is far more frequent in boy babies than in girls. It is highly probable that two factors operate to produce the active symptoms: 1. A congenital hypertrophy of the sphincter fibers of the pylorus. (Such hypertrophy has been observed in a seven months fetus.) 2. Spasm of the pyloric sphincter produced by irritating food or possibly by excessive acidity of the gastric contents. These views of causation explain why symptoms may remain

absent for several weeks even though the hypertrophy is congenital. Cautley contends, and there is much clinical evidence to support him, that there are two conditions that may closely resemble one another: 1. True hypertrophy of the pylorus of congenital origin. 2. Spasmodic stricture of the pylorus. He believes that most of the cases that recover without surgical intervention are of the latter character.

**Symptoms.**—The symptoms may appear immediately after birth, or when the child is from one to five weeks old. The child is usually restless, desiring drink, but attempts at swallowing are soon followed by vomiting. Occasionally at first the vomiting occurs at long intervals, but rapidly increases in frequency until it follows all attempts at swallowing. The vomited matter consists of the materials swallowed, together with a large amount of mucus, but is almost never bile-stained. There is hyperchlorhydria. The tongue is clean. The bowels are obstinately constipated, and the amount of urine is often lessened. Pyloric tumor is detected in at least 50% of cases. Peristaltic waves are observed in the epigastric region. Often the lower abdomen lacks prominence, in striking contrast to the epigastrium. Nutrition rapidly fails, and the child usually dies of inanition.

**Treatment.**—The first indication in the treatment is the careful regulation of the feeding. The amount of food should be small, and must be given at frequent regular intervals. It should be of such a character as to be easily and quickly digested. No refuse food should be allowed to accumulate, for it must be remembered that pyloric stenosis is an occasional cause of gastric dilatation, and to prevent this the stomach should be emptied by irrigation and washed out once or twice daily. Enteroclysis is of vast service, tending to lessen vomiting, and also serving to supply the lack of body fluid. Hypodermoclysis may be necessary in some cases. Peptonized foods and buttermilk have served us best; but the diet must be adapted to the individual baby. If no food can be retained and nutrition fails, surgical methods must be considered, and not too long delayed. Pylorectomy and anterior gastro-enterostomy are the procedures followed.

#### GASTRIC AND DUODENAL ULCERS

In infancy and early childhood these morbid processes are rarely seen, though in anemic girls, at the age of puberty, they are fairly common occurrences. Soltau Fenwick collected 19 cases, the youngest (a fatal case of hematemesis), being but thirty hours old. His cases include all varieties of ulcerations. Fischl without pretense of an exhaustive study collected 21 cases of the simple perforating variety.

**Etiology.**—The following types of ulceration are described by Holt. The cause is suggested in each instance.

1. Ulcers in the new-born. These are noted in cases of hemorrhagic disease of the new-born. They are usually follicular and small, resembling follicular ulcerations of the colon.

2. Ulcers resulting from acute gastritis.

3. Tuberculous ulcers. (These are better described as ulcers in tuberculous subjects.) In autopsies upon 119 tuberculous subjects, Holt met gastric ulcers five times. In the ulcerative lesions of three of these cases, tubercle bacilli were found.

4. Simple perforating ulcer.

To this etiologic table should certainly be appended:

5. Ulcers of the stomach and duodenum observed after severe burns.

6. Nor should typhoid fever, diphtheria, noma, scarlet fever, pneumonia, septicemia, etc., be omitted from the list of contributory causes.

Ulcers may occur in other cachetic states than tuberculosis, and their occurrence is favored by various anemic states.

**Pathology.**—Ulcers may present a clean, punched-out appearance, without inflammatory margins (Fenwick). Such may be observed in cases of bad burns. The follicular ulcerations of early life, or those appearing in the infectious diseases of later childhood, may appear dependent upon inflammatory causes (gastritis of toxic origin); though, in other cases, there is ample evidence of hemorrhage, thrombosis, or both ("hemorrhagic erosion"). Blood may be found within the gastric viscus—sometimes in considerable quantity—and in the hemorrhagic disease, hemorrhages may have taken place into other organs (the suprarenal capsules, etc.). The ulcers of the afore-mentioned types are found more often toward the cardiac end of the stomach and along the greater curvature. Such is not the case with the rarer chronic ulcers. They are more likely to occupy the classic situations where they occur in later life, viz., the lesser curvature toward the pylorus. Fenwick states that several factors here cooperate to retard healing: 1. This portion of the viscus receives a smaller blood supply—postmortem injections revealing this fact graphically. 2. The activity of the organ in this situation. One may compare its effect on the healing process to the influence of joint activity upon skin lesions in the neighborhood of the joint. 3. The firm attachment of the mucous membrane to the submucous tissues. The more chronic ulcers of this type exhibit the typical "terraced appearance" and thickened inflammatory margins. Peritoneal adhe-

sions may exist between the liver, pancreas or other organs. In case of a complete perforation, the peritoneal sac may be distended with gas alone or it may contain gastric contents and blood. In one case a fistulous tract had passed through the diaphragm and communicated with a pyopneumothorax of the left chest (Leith).

**Symptoms.**—The symptomatology and clinical course of gastric ulcers in early life depend upon the cause and the type of the ulceration. Age also makes a difference, as young children may yield practically no subjective symptomatology. In infancy (in the hemorrhagic disease, etc.), hematemesis may be the only symptom that makes one suspect ulceration. The suspicion may not be verified, even postmortem, for vomiting of blood may be dependent on many many other causes. Tuberculous subjects may yield absolutely no evidence of their gastric ulcers.

Simple perforating ulcers ("peptic") may give rise to much the same phenomena that they occasion in later years, viz: Pain in the epigastrium or back; various dyspeptic symptoms with acid eructations; tenderness over the ensiform cartilage (Fenwick) or between the ninth and tenth dorsal vertebræ (Fischer); vomiting, sometimes of bright blood, at others of darker blood in smaller amounts ("coffee ground"); hyperchlorhydria, constipation, and anemia. On the whole, the younger the child, the less typical the picture. Nevertheless, the character of the pain should place one on guard. It becomes immediately worse when food is ingested, and continues to grow more severe for several hours thereafter. Ofttimes it is not relieved until vomiting takes place. Vomiting—particularly hematemesis—furnishes valuable positive evidence; but its absence should not prevent one from suspecting the dangerous condition present. An examination of the gastric content should always be made. Occult blood may be recognized in vomit or stool. The existence of hyperchlorhydria represents strong confirmatory evidence.

Perforation is often proclaimed by a sudden agonizing pain followed rapidly by symptoms of collapse. The recti muscles become powerfully contracted (rigid) and abdominal palpation becomes almost an impossibility. The abdomen is retracted and exquisitely tender. This condition is soon replaced by abdominal distention, with obliteration of the hepatic and splenic dulness, leukocytosis, and other evidences of peritonitis.

**Diagnosis.**—As we have just intimated, under some conditions, the diagnosis of gastric ulcer is almost an impossibility. But, when a simple perforating ulcer does occur in an anemic girl, or where

symptoms appear after a severe burn, the condition should be diagnosed. In younger children, it may not present so typical a feature; but on the other hand, there are not so many conditions with which it may be confounded as in later life (gall stones, carcinoma, etc.). It should be carefully differentiated from appendicitis in early life. The symptoms and signs of perforation should also be well known. It may be possible in some cases to administer to the child a mixture of a suitable dose of bismuth in milk and photograph the ulcer by the X-ray.

**Prognosis.**—This depends upon the cause and upon the kind of lesion. In the main, the existence of a gastric or duodenal ulcer should spell gravity. The smaller ulcers occur in serious conditions, while the simple perforative lesions are potentially most dangerous. Ulcers of the latter type probably always take a considerable time to heal.

**Treatment.**—In the majority of the ulcerations described as occurring in the early years, the treatment is the treatment of the underlying state and is described under the appropriate heading. The initial treatment of the simple perforating ulcers may be expressed in one word—rest—and that means rest for the body in bed, and rest for the stomach in abstinence from food. Rectal feeding should be tried with the following facts before us: 1. No one can tell in advance how much absorption will take place. In Edsall's experiments, the results were most disappointing. On the other hand every clinician of experience has seen it act well. 2. The food should be fully peptonized. 3. The food should be concentrated and the amount injected small (not more than from 2 to 4 ounces). 4. The toleration of the lower bowel must be carefully tested, and if irritable, the interval between injections should be long. 5. It is best to make the injection as high as possible. When mouth feeding is resumed, it should be with the greatest care. This is a mooted field in dietetics, but we prefer to begin with small amounts of peptonized milk, given at rather frequent intervals. Our next advance is to the proteid foods; our next to the green vegetables. Carbohydrates and fatty foods we do not give until convalescence is well advanced. Taken all in all, the drug that has proved most useful in our experience has been bismuth given in large doses and at frequent intervals. Alkalies in large doses (usually bicarbonate of soda) has given much relief from the terrible burning pain. We always give them a thorough trial before resorting to morphin. Orthoform is also said to be very useful for the same purpose. The application of the Preissnitz compress often yields very good results when the "burning" sensation is severe. Morphin must be

employed at times, so must cocain. Theoretic objections may be raised, however, against the use of both drugs.

Hemorrhage, if severe, may be combated by the use of adrenalin internally (really locally) and of calcium salts. Despite the fact that more recent studies have seemed to show that the calcium salts do not shorten the coagulation time, we are both convinced of their value in hemorrhagic conditions. The subcutaneous injection of gelatine, may also be tried. Should the patient suffer a great loss of blood, enteroclyses and hypodermoclyses of normal salt solution are indicated.

If the diagnosis of gastric ulcer has been made, and there is evidence pointing toward a perforation, operation should not be delayed. Even though this accident has not taken place, the operator may be able to excise the offending lesion.

### GASTRALGIA

**Definition.**—The term gastralgia is applied to a sudden, severe attack of pain in the gastric region unaccompanied by inflammation.

**Etiology.**—The condition may arise from exposure to cold by taking cold drinks, especially when the child is overheated, or occasionally by getting the feet wet. Not infrequently it appears as a form of neuralgia. Holt states that it is common in children affected with malaria, especially at the onset of the attack. The amount of pain may be slight, or may be so severe as to cause faintness or marked prostration. No inflammatory symptoms accompany the pain.

**Treatment.**—The patient should be put at rest in bed, and counterirritation over the stomach applied by means of a turpentine stupe, mustard plaster, or hot-water bag. Internally should be given moderate quantities of hot water containing five drops of spirits of chloroform, Hoffmann's anodyne, brandy, whisky, or gin. If the pain is severe enough to cause prostration, heat should be applied to the body and all food withheld during the attack. Recurrent cases are best treated by the use of arsenic in the form of Fowler's solution. Attention should also be directed to the patient's general health and to careful regulation of the digestion.

### DILATATION OF THE STOMACH

Frequently the stomach becomes dilated as the result of long-continued chronic catarrh of its mucous membrane. The condition

is most commonly found in artificially fed rachitic or anemic children; for this disease may diminish the tone of both voluntary and unstriated muscular tissues. The walls of the hollow viscus yield, and the support furnished by the abdominal muscles is lessened. Occasionally, though rarely, it arises from acute gastro-enteritis and cholera infantum (acute conditions seldom cause dilatation). It is also seen as a secondary consequence of congenital stenosis of the pylorus or obstruction of the duodenum and ileum. (See article on this subject.)

**Pathology.**—As a result of weakness and insufficiency of the digestive fluids decomposition of the casein of milk and all starchy elements takes place, resulting in the formation of large quantities of gas. These keep the weakened muscular coats continually on the stretch, and, as a result, atrophy of the muscular fibers and glands takes place; such a stomach is never entirely emptied, but always contains more or less decomposing food and mucus, from which often results an autointoxication. The size of the stomach in this condition is sometimes very great. Thus, Henschel records the case of the stomach of an infant two weeks old the capacity of which was 190 c.c., the normal capacity being 70 c.c., and he also gives an account of the stomach of an infant of three months whose gastric capacity was 485 c.c., the normal capacity being 150 c.c. Other cases showing the great increase in size of the stomach owing to dilatation are reported by the same author and others.

**Symptoms.**—The symptoms of gastric dilatation are frequently vague, and it is not uncommon for the condition to be discovered only at the autopsy. Such symptoms as chronic dyspepsia, regularly occurring discomfort after taking food, and habitual vomiting after meals may lead us to suspect the condition. The child if old enough complains of pain after eating for some days, and then suddenly vomits large quantities of partially digested fermented curds or the remains of other food taken during this time. The tongue is heavily coated, there is frequently constipation, and the child shows symptoms of general nutritional failure. In the epigastric region inspection reveals an undue prominence. Peristaltic waves may also be noted. Palpation or “flipping” may cause “the stomach to stand up.” Percussion is of some value, though one must carefully distinguish between the note yielded by the gastric viscus and that elicited over the transverse colon. Pfahler and others have also secured brilliant diagnostic results by placing kefir and bismuth solutions in the stomach, and then pursuing skiagraphic studies. In skilful hands the gastrodiaaphane may be employed in older children.

**Diagnosis.**—Owing to the obscurity of the symptoms the diagnosis may not be easy. It is said that a splashing sound may occasionally be produced by shaking the child gently, but this sound can only be heard when the stomach is full of fluid. The careful pursuance of the diagnostic procedures just mentioned will usually repay the examiner. The condition should be carefully distinguished from the more common gastroptosis.

**Treatment.**—The treatment is that of chronic gastric indigestion. The food should be carefully modified and given in small quantities at regular intervals. Irrigation of the stomach is especially indicated. Strychnin or nux vomica, arsenic, and the mineral acids, particularly hydrochloric, are the chief remedies to be employed. Such children should be kept in the best of hygienic surroundings and allowed a life in the open air as much as possible. Abdominal massage is a useful measure. For older children so are the abdominal exercises of the Delsarte mattress work. It is our custom to reinforce the support of the abdominal muscles with a firm elastic binder.

### GASTROPTOSIS

Gastroptosis and visceroptosis are not known to be common conditions in infancy and early childhood. In later childhood and in adolescence they become common enough. Still, one cannot see many rachitic babies, and fail to note how lax the abdominal walls become. Both the voluntary muscles of abdominal wall and the unstriped muscle tissue of the hollow viscera suffer, and the typical "fat-belly" results. And having noted these results of innutrition, and having pondered over their causes, one cannot resist the impression that some cases of visceroptosis really originate in infancy.

## DISEASES OF THE INTESTINES

### MALFORMATIONS OF THE INTESTINAL TRACT

#### Stenosis and Atresia

Narrowing or closure may occur at any part of the intestinal tract. The causes may be divided into congenital and acquired. The congenital form is usually due to the formation of cicatrices resulting from intestinal ulcer occurring during intra-uterine life, or to fetal volvulus. The condition may also arise from the formation of peri-



upward in search of the rectal opening. In doing this a staff should be placed in the bladder as a guide. Should this fail, Rotch advises that Littré's operation be performed. This consists in opening the sigmoid flexure in the inguinal region and making an artificial anus; or an attempt may be made to cut through the sacrum and make an opening into the gut at this point. Quite recently one of us advised operation in a baby thirty-six hours old. A rectal examination was made, and a much distended coil of bowel was detected high up. The operator brought this down, stitched it to the anal opening and incised the distended gut, but little relief was afforded and the baby died that night. An autopsy was performed by McKee and yielded most interesting finds. A loop of jejunum had been incised at the operation. The whole large intestine looked like a small fibrous cord. Many distended loops of small bowel were found and there were a number of points of stenosis in the caliber of the small intestine. (See Illustration.)

### CONGENITAL DILATATION OF THE COLON

**Synonyms.**—HIRSCHSPRUNG'S DISEASE; CONGENITAL IDIOPATHIC DILATATION OF THE COLON

**Definition.**—This a congenital condition of obscure origin in which the colon is found lengthened, dilated to a great extent or exhibiting true hypertrophy of its dilated walls. Concetti distinguishes three varieties: mycrocolia (some lengthening), ectocolia (ectasia of a small or large portion) and megalocolia (great enlargement in the intestinal caliber and thickening of the walls).

**Etiology.**—Some patients are unquestionably born with this condition, and Hirschsprung and others would limit the term to include these cases. Their origin is very obscure. Hypertrophies have been found in other parts of the intestinal canal, in one case, curiously enough, where there was duodenal stenosis. In another group of the cases, obstruction below the dilated or hypertrophied area would seem to have played an important etiologic rôle. Such obstruction is usually found in the unduly elongated and convoluted sigmoid flexure.

**Pathology.**—There is little to add to what has been said. Microscopically and even to the naked eye, it is clear that the muscular coat of the bowel is the one that is thickened (hypertrophy). The affected portion of the bowel may attain enormous size. Ulcers may occur

in the mucous membrane and lead to septic involvement of the sub-mucous layer. Abscesses have here been found.

**Symptoms.**—Two classes of cases are recognized clinically (Foehl), though some authorities (Mya, Griffith, etc.) would exclude cases dependent upon obstruction below the part dilated: 1. The cases in which symptoms are manifested soon after birth. 2. Cases in which symptoms do not appear until a later period (weaning time). Quite characteristic is the occurrence of a large number of liquid stools, after a period of constipation, with almost immediate relief of the symptoms outlined. The same result may follow abdominal massage combined with rectal manipulations or large enemata. The symptoms soon recur, however. Briefly, in both varieties, the following symptoms are noted: Obstinate constipation, often unrelieved by laxatives, suppositories and enemata; marked ballooning of the abdomen (mayhap to a degree seen in no other condition); dilatation of the superficial abdominal veins; dyspnea from pressure on the diaphragm; intoxications of intestinal origin; possibly severe nervous symptoms (meningitis) and death.

**Diagnosis.**—If the condition is known to the observer, there should be no difficulty in its clinical recognition. It must be distinguished from congenital intestinal anomalies of other varieties and from abdominal growths (renal sarcomata). The history, the symptoms and the physical examinations should yield positive results.

**Prognosis.**—This is usually grave, though some of these patients have lived to adult life. Life may be prolonged, or in some instances saved, by appropriate treatment.

**Treatment.**—If enemata are ineffectual, and large enemata (under low pressure) should be tried, enemata, combined with systematic abdominal massage should be employed. Purgatives, in advanced cases, are contraindicated. Massage of the abdomen while the well lubricated finger is in the rectum may succeed better than the afore-mentioned measures. Electric treatment (usually faradic) should be given a thorough trial in these cases. An electrode is sometimes introduced into the bowel after a large saline enema has been given. The tincture of nux vomica or strychnine sulphate should be administered in rather full dosage in these cases. Eserine is employed by certain authorities. Where symptoms are very urgent, it has been suggested that the bowel be punctured. To us this seems a dangerous measure and any case seeming to demand it had better be operated upon by a skilful surgeon. Several operations have been devised and pursued.

## ACUTE ENTERITIS

**Synonyms.**—ACUTE INTESTINAL INDIGESTION; ACUTE CATARRHAL ENTERITIS; SIMPLE DIARRHEA; MECHANICAL DIARRHEA

**Etiology.**—Food given in too large quantities or of a sort not adapted to the age and condition of the child, irregular feeding, the use of a dirty nursing-bottle, the too early and frequent use of table foods, and bad hygiene may be accepted as the most usual causes of acute enteritis. Any or all of these causes may increase the severity of the attacks when they occur during the period of dentition, at which time the entire nervous system of the child is readily influenced by external causes, and the digestive tract is in a condition which may be termed hyperesthetic. Another predisposing cause may be sudden changes of the temperature, especially a rapid change from cool weather to hot.

**Symptoms.**—The attack of enteritis usually begins with an increased number of stools, averaging anywhere from five to twenty a day. The bowel movements, which for the first two or three evacuations are normal in color and consistency, rapidly change to liquid or soft unformed masses of a greenish or yellowish hue. All the bowel movements contain curds and more or less mucus. The evacuations are preceded by pain and tenesmus. In a few hours the child suffers some loss of flesh, which is particularly manifest in the face and limbs. When the attack is of short duration, the abdomen may be painless on pressure, but as a general rule the child will complain of some pain in this region on palpation in a few hours if the attack continues. Vomiting may or may not be present. Occasionally the disease is ushered in by convulsions, but this symptom shows a severe degree of intestinal irritation or poisoning. Thirst is nearly always a prominent feature. More or less distension of the abdomen is usually found during the first hours of the disease. The pulse, although increased in frequency during the attacks of pain, is not usually much above normal. If fever is present, it is rarely constant, but some irregular increase of temperature is generally observed.

**Diagnosis.**—This may be made from the history (particularly of dietetic indiscretion), the symptoms and the examination of the stools. The condition must be distinguished from the various forms of gastro-enteric infection.

**Prognosis.**—The prognosis is favorable under proper treatment. The principal danger consists in allowing the irritating masses of

food to remain in the intestines long enough to set up a condition of chronic inflammation.

**Treatment.**—When the case is seen early, a dose or two of a dram of castor oil will, in many cases, remove the irritating masses of undigested foods. When the child is old enough, this may be given in the form of a soft capsule, each one containing from five to ten drops of oil. In young children the remedy may be given in emulsion or sandwiched between two layers of five or ten drops of whisky or brandy, or the oil may be introduced into the rectum. If calomel is indicated in these cases it may be either given alone or preferably in combination with salol or bismuth. Where calomel is used in these cases, the drug acts not only by its laxative effect, but also indirectly by increasing the flow of bile into the intestines. An effective mixture used by us in hundreds of dispensary cases consists of equal parts of lime-water and cinnamon water, of which two drams are given every hour or two, and often three grains of bismuth subgallate are added. When the amount of pain is very great, the aromatic syrup of rhubarb, in doses of from a dram to half an ounce, will be found useful. The use of opiates in cases of acute indigestion is not to be encouraged, nor are astringents to be given until the intestine has been thoroughly cleansed of the irritating cause of the attack. In most cases the thorough evacuation of the intestines should be encouraged by copious enemata of boiled water containing a small amount of Castile soap, ten or fifteen grains of bismuth subnitrate, or 6 per cent. of sodium chlorid.

The diet is a matter of great consideration. During the summer season, if the child has been fed by the bottle, all milk, or preparations containing it, should be stopped until the bowel movements regain their normal character. It is so easy to implant an infection under these conditions. During the first six or eight hours of the time during which milk diet is withheld the patient should receive small quantities of sterilized water containing fifteen or twenty drops of brandy every two or three hours. After the initial starvation the carbohydrate foods—barley water or dextrinized barley jelly—are the best foods. If milk has been discontinued, it is our custom to resume it with a weak formula, and peptonize the food in the bargain. A good rule is: Second twenty-four, we use a formula one-fourth of the former strength; third day, one-half strength; fourth day, two-thirds strength; fifth day, three-fourths strength, etc. When milk cannot be resumed so soon, an excellent combination is: Two parts of dextrinized barley jelly and

one part of albumin water. Both albumin water and beef juice may act badly, however.

In twenty-four hours after the stools have resumed their normal color the child may be gradually returned to its usual diet; it is well, however, to give the patient a drop or two of tincture of *nux vomica* three or four times a day before feeding, in order to stimulate the assimilative action of the intestines.

### CHRONIC ENTERITIS

**Synonyms.**—CHRONIC INTESTINAL INDIGESTION; CHRONIC CATARRHAL ENTERITIS; CHRONIC IRRITATIVE DIARRHEA;  
CHRONIC INTESTINAL CATARRH

**Etiology.**—The causes of chronic intestinal catarrh are continuation or return of a series of attacks of acute intestinal indigestion; the continued use of improper foods, especially at the period of dentition. Chronic enteritis may follow any of the infectious diseases, exposure to cold and wet, or bad hygienic surroundings. Chronic intestinal catarrh is fully as common in winter as in summer, although the type seen in the hot months is severer and runs its course quicker than in cold weather. The disease is most commonly seen from the third month to the end of the second year, and is much more frequent in artificially fed children than in those fed from the breast; indeed, providing that syphilis, rickets, or tuberculosis is not present, it is somewhat rare to find chronic intestinal catarrh in breast-fed babies. The majority of cases are caused by attempts at feeding a nursing child on a badly prepared artificial food, especially condensed milk or cow's milk in improper proportions.

**Symptoms.**—The diarrhea that has been present during an acute attack continues, or returns after a period of cessation. The bowel movements are watery, or they may occasionally change to semi-formed masses of a grayish-white, putty-like hue and consistence. They contain undigested food and much mucus. The amount of mucus may be so great as to suggest the old designation mucous disease. Green stools are common, and these may last for quite a considerable period, particularly at the beginning of each exacerbation of the disease. The number of the stools will usually average from four to ten a day, and are preceded and accompanied by considerable pain and tenesmus. Lientery may be manifested, the taking of food promptly inducing a stool. The disease is subject to distinct exacerbations, and it is not infrequent for the medical attendant to imagine

that he has the case well under control and later to find, to his discouragement, that the disease has returned with renewed vigor. These cases imperatively require careful attention and patience, not only on the part of the physician, but also on the part of those having charge of the little patient. While a moderate loss of weight is sure to appear finally, and, indeed, may become very great, it is not infrequent to see patients stand the continued drain on the system from diarrhea and lack of assimilation of food remarkably well. The loss of flesh is particularly noticed in the limbs and face, the former losing their roundness of shape and firmness to the touch and the latter its characteristic plumpness and happy expression. The facial expression of these children is remarkably old, thin, and tired. The fontanel is depressed, and the lower part of the face assumes an angular shape which is eminently characteristic. The abdomen is either depressed or considerably swollen; the child is fretful, cries a great deal, and is extremely restless during sleep, young children tossing and frequently crying out, and older children exhibiting the group of symptoms known as night-terrors. The appetite is capricious, sometimes almost lost and at other times the child manifests a ravenous desire for food. Occasional attacks of constipation are not infrequent. The tongue is red, dry, or may be covered by a brownish or yellowish coat. Relaxation and prolapse of the rectum are not uncommon. The skin is often dry and scurfy. The temperature is generally normal, although short periods of slight pyrexia are not infrequently seen. Many of these cases run their course without gastric complications.

**Diagnosis.**—Although the diagnosis of chronic intestinal catarrh is by no means difficult, cases presenting the before-mentioned symptoms should be carefully examined before a positive opinion is given. The disease may occasionally be confounded with general tuberculosis with intestinal complications; but in tubercular affections we have the regular daily rise and fall of temperature, the large offensive watery bowel movements, and probably evidences of tubercle in other parts of the body, especially the glands and lungs.

**Prognosis.**—The prognosis depends in great measure upon the duration of the disease, the general constitutional condition of the child, and the treatment it receives. The coexistence of other diseases, such as syphilis, tuberculosis, rachitis, etc., makes the prognosis more unfavorable. Amid good hygienic surroundings, with careful regulation of the diet and the administration of proper remedies, the prognosis is generally favorable, although the duration of the condition is usually rather tedious.

**Treatment.**—The success of the treatment of chronic gastrointestinal catarrh depends far more upon the food and general surroundings of the child, its place of living, its bathing, clothes, etc., than upon the administration of any drugs. In young children nursing from the breast the composition of the milk should be carefully investigated. The existence of pregnancy, prolonged hard work, nervous excitability, prolonged lactation, and many other causes may so alter the constitution of the mother's or nurse's milk as to produce chronic dyspepsia in the child. In such cases, of course, the diet should be regulated, and, if possible, another wet-nurse should be substituted, or, when the child is nursing from the mother's breast, it should be provided either with a wet-nurse or placed on artificial diet. It is extremely difficult to lay down any fixed rule for the diet of these children. The child should be nourished on that class of foods which he most easily digests and assimilates. In many cases the stools will contain large undigested masses of casein or fat, or they may be highly acid; especially is this the case when the child has been fed on foods rich in sugar. In this class of cases all milk should be prohibited and the child fed for a considerable period on beef peptonoids, panopeptone, meat-extracts, albumin-water, broths, or other species of proteid foods. When the stools are alkaline in reaction, frothy, or putrid, a moderate diet of starchy or dextrinized foods or foods containing some sugar will often answer admirably. It must be borne in mind, however, that, as a general rule, no child under eight months of age will thrive continuously on a diet of starch. Modified milk containing a low proportion of sugar and fat and a high percentage of proteids is often of great use, and to this should be added 10 or 15 per cent., by bulk, of lime-water. Peptonized milk, either alone or combined with arrow-root, is sometimes of service, but this diet cannot be kept up for long. In older children the diet is a matter of the greatest importance, but it is often very difficult for the physician to control and direct it in a satisfactory manner. All irregular meals, highly seasoned foods, candies, and most of the sweet cakes must be strictly prohibited. It is far better that the child should occasionally receive too little food than that it should eat food of improper quality or in quantities too large for its temporary capacity. The following diet-list has been recommended by Starr for these cases, this, however, being subject to modifications and frequent changes.

*Breakfast.*—At 8 A.M., a teacupful of bread and milk; the milk should be rendered alkaline by the addition of lime-water. In not very severe cases a lightly boiled or poached egg, with one or two slices

of bread made from unbolted flour, or stale bread, with butter, may be substituted for the bread and milk.

*Dinner.*—At noon a lean broiled mutton chop or a piece of under-done tender roast beef; such vegetables as cauliflower or a small quantity of well-roasted potato (not new) may be added. A small quantity of bread well toasted or Zwieback; occasionally a half an ounce of good sherry well diluted.

At 4 P.M. about twelve ounces of fresh milk, rendered alkaline as previously mentioned.

At 7 P.M. supper: a cup of beef-tea or mutton broth.

A diet-list such as the preceding may be continued as long as the child does well. Whenever a relapse occurs, a return to a strict diet of modified milk, or, better, beef-juice, beef-tea, or chicken or mutton broth, given in small quantities at regular intervals, and this plain diet continued until the bowel movements have become normal in color and have been free from mucus for a number of days. The question of the climate and general surroundings of the child is one for earnest consideration. Children with intestinal disease, particularly of a chronic form, do much better in the country than in the city, but they improve more rapidly at the seashore than anywhere else. It is also of importance that such children be kept in the air, and they should be encouraged to play out-of-doors. Play should be interrupted at least once (better twice) a day, however, and an hour's rest obliged. Even in winter, providing the weather is not too windy or damp, they should be kept in the open air for a considerable portion of each day. The clothing should be of wool, and so constructed as to impede as little as possible the free movement of the limbs.

**Medicinal Treatment.**—The medicinal treatment should be directed first toward thoroughly clearing the intestinal tract of the irritating cause of the disease, and, secondly, stimulating the digestive and assimilative powers. For the first, laxative doses of castor oil are occasionally of great use. Calomel, also, is an exceedingly useful drug for this purpose. It is best given in small and frequently repeated doses, and may be combined advantageously with salol or bicarbonate or phosphate of sodium. Beta-naphthol bismuth, in doses of from one to five grains repeated every few hours, has been highly recommended by Dr. Lewis Fischer and others. The following prescription is recommended by Rotch:

Podophyllin.....	1 grain
Alcohol.....	1 dram.

Give from three to five drops, according to the age of the child,

repeated morning and evening, the dose to be lessened if it causes more than two discharges a day. Tonics, and particularly arsenic and nuxvomica, should be given. From one to five drops of the tincture of nuxvomica given immediately after taking food forms an excellent digestive tonic. Of the intestinal astringents, bismuth is probably the best: doses of from five to ten grains of the subgallate, or subnitrate, given either alone or in combination with salol, give excellent results. Smaller doses of bismuth salicylate may also be employed. In many cases bismuth may be advantageously administered by means of enemata applied to the lower bowel. Opiates, as a rule, are distinctly contraindicated. Probably one of the most successful methods of treatment is by copious enemata of warm sterilized water or normal salt solution; the latter has been recommended by Fischer, of New York. From three to four quarts of water should be used at each irrigation. They should be given from a fountain syringe, to which is attached a large-sized soft catheter having more than one opening. The child should lie on the nurse's lap, either on its back or in what we have found to be better positions—namely, on the abdomen or left side. The syringe should be held about three feet above the floor, and the water should be allowed to flow in and out again. It is often necessary to flush out the intestines thoroughly with either plain boiled water or normal salt solution, in order to clear them before administering the enema containing the intestinal antiseptic or astringent. Probably the best antiseptic to be given in this way is a combination of subnitrate of bismuth and salol, or beta-naphthol bismuth. They should be administered in a half pint or so of water, allowed to flow into the intestine, and remain there.

When anemia is a complication of this disease, and especially if it persists after the recovery of the child, some of the numerous preparations of iron are indicated. While, as a rule, cod-liver oil or any of the vegetable oils are contraindicated when administered by the mouth, yet not infrequently we find excellent results following inunctions of these applied to the abdomen or over the body generally. The benefit of these inunctions is undoubtedly increased when accompanied by massage.

#### ACUTE GASTRO-ENTERIC INFECTION

**Synonyms.**—ACUTE MILK INFECTION; CHOLERA INFANTUM; THE TERM "CHOLERIFORM" IS SOMETIMES USED

The disease here described as acute gastro-enteric infection is that form of acute gastro-intestinal poisoning usually produced by those

bacterial toxins peculiar to milk and foods containing milk. Such infections, however, may result from other foods than milk. True cholera infantum is becoming a very rare disease.

Acute gastro-enteric infection is a disease almost peculiar to the warmer months, and is found usually in children fed on an artificial diet of foods containing milk. It occurs very rarely in children fed upon breast milk, although occasionally such cases have been reported.

**Etiology.**—The specific poison, bacterial or chemic, producing cholera infantum has not yet been isolated. Probably there are several bacteriologic causes. In the healthy nursing child two forms of bacteria are constantly found in the intestinal tract; these are the *bacillus lactis aërogenes* and the *bacterium coli communis*. These are sometimes called the “obligatory milk feces” bacteria. According to the researches of Escherich, Baginsky, Booker, and others, the upper part of the duodenum is quite free from micro-organisms, while the lower part of the small intestines contains considerable numbers of the *bacillus lactis aërogenes*. The *bacillus coli communis* has been found chiefly in the lower part of the ileum, and still more abundantly throughout the entire length of the colon. Whether or not the poison is due to a sudden increase in number of these bacteria or their toxins produced partly by the continued high temperature of the summer and aided by a fermentation of artificial foods in the intestines, or whether it is due to some specific microbes which have their existences only during the warmer months, is still an unsettled question. Certain it is, however, that acute gastro-enteric infection is almost solely found during the summer months. It is a disease much commoner in cities than in the country, and the number of cases increase during the prevalence of a temperature above 70° F. (21.1° C.). The children of the poor are oftener attacked than those in better circumstances, and, as before stated, it is found almost entirely in children fed on artificial foods containing milk or on unsterilized cow’s milk, and some of the very worst cases that we have seen have been among infants fed on condensed milk. It appears most frequently from the third month to the end of the second year. Although the toxic bacteria are generally introduced into the system in the manner before stated, they may enter in other ways, as by the anus or by the mouth from the nipple of a mother or nurse whose habits are uncleanly. An infant may infect itself from its own fingers, which in many cases are far from clean.

**Pathology.**—In infants dying after an acute attack of milk infection of short duration, we find a very considerable amount of emaciation, much more marked in the face, however, than in the body; the cheeks

and eyes are sunken, and the fontanel is depressed. If the disease has lasted for some days, the loss of bodily weight is extreme; the limbs lose their rounded shape, and the skin covering the upper part of the thighs is loose and hangs in folds. The face has entirely lost its plumpness and shows extreme emaciation. Upon opening the body, examination will show minute hemorrhages upon the surfaces of the lungs, with evidences of hypostatic congestion at their bases. The same minute hemorrhages are found in the heart, which is usually in a state of diastole, its cavity being filled with blood. The mucous membranes of the stomach and intestines are in a condition of capillary congestion, with small hemorrhagic patches scattered here and there. The contents of the intestines are liquid. Peyer's patches and the solitary glands are swollen. There may be marked congestion of the mucous membrane of the entire large intestine, this being greatest in the cecum and descending colon, though in other cases the mucous membrane of the bowel is blanched. Ulceration is rarely observed in this condition, as time is required for their production. As a safe generalization, one may say: The morbid anatomy of these gastro-enteric infections does not bear a direct relationship to severity; rather, it depends on the time that the disease has lasted. In many instances catarrhal pneumonia will complicate the later stages of the disease, and in these cases solidification will be found at the bases of the lungs. According to Ashby and Wright, a microscopic examination of the mucous membrane of the intestines shows a general distention of the network of their capillaries and an exudation of leukocytes. This condition exists in the mucous membrane itself, the submucosa, the villi, and between the tubules and crypts of Lieberkühn. The central portions of the solitary glands are softened, or the softened portion having been discharged, the remains of the glands appear as sharply cut ulcers.

Examination of the brain shows no specific lesion; occasionally the sinuses are found distended with blood, or, on the other hand, a condition of cerebral anemia may exist. Ashby and Wright mention one case in which meningitis was present, but these instances are extremely rare.

**Symptoms.**—No matter how the poison enters the system, the symptoms of acute gastro-enteric infection are nearly always the same, unless indeed, we except the now rare cases of cholera infantum. Occasionally a mild diarrhea may precede by a few days the sudden onset of the acute indications. During this time the stools are more frequent than the normal and are green. There may be some vomiting

after taking food. It is very questionable, however, whether this preliminary diarrhea is part of the true attack of cholera infantum; on the other hand, the diarrheas of the summer season so readily pass from one form into another. Very frequently the disease begins suddenly at a time when the child is in perfect health. The first symptoms are, then, vomiting and purging. The vomiting is intense and continuous. The vomited matter is first composed of the gastric contents, later of watery detritus, and, finally, after there is nothing left in the stomach to be thrown off, the attempts at vomiting continue. The child has nausea of the most intense character. The vomiting is increased by the taking of food or drink—in fact, anything put into the stomach is immediately expelled. During these paroxysms the child becomes pale, the lips are blue, a dark line is seen around the mouth, and the entire body is covered with cold, clammy sweat. The evacuations of the bowels occur more and more frequently, until they are practically continuous. Their character changes with great rapidity from the normal yellow movement of the infant to a thin, green, spinach-like discharge, and finally, in the true cases of cholera infantum, an almost continual expulsion of large quantities of water mixed with shreds of mucus, which give them somewhat the appearance of rice-water. These discharges have a characteristic musty odor, are acid in reaction, and are composed of serum mixed with epithelial cells and swarming with bacteria. The abdomen in the earlier stages of the disease may be slightly distended and soft, but as the diarrhea continues it becomes retracted. A marked difference exists in the temperature of the surface of the body and of the interior. Occasionally during the first few hours the surface temperature may be above 98.6° F. (37° C.), but in the majority of cases it does not rise above the normal point. In the algid state of extreme depression it is distinctly subnormal. The rectal temperature will be found to be anywhere between 103° and 107° F. (39.4° to 41.6° C.) early in the disease, though in cholera infantum, it may later sink to the normal or subnormal stage. Very weak babies may perish without any rise in temperature. Such cases are not rarely seen in institutions. The average duration of the disease is from one to three days, although we have seen cases in which the entire attack, from the first symptoms until the death of the child, has lasted but six hours. The loss of flesh is appalling, the child frequently changing in the course of a few hours from a rosy, plump baby to a mere skeleton covered with skin. There is probably no other disease, with the exception of Asiatic cholera, in which the emaciation is so extreme in so short a time.

This rapid decline in bodily weight is due to the rapid loss of fluids (anhydremia). As the disease progresses the respirations become shallow and jerky, and the child passes into a state of coma, convulsions, or, rarely, delirium. It is not unusual to find a short interval during which there is a lull in the symptoms. At this point in the disease the child may begin to improve, but much more commonly this interval is quickly followed by an increase in the symptoms of profound nervous depression, the child passing into a state of coma, followed by death. When the amount of poison is originally very great, the patient may become comatose in a very few hours. These cases are practically hopeless. During the entire attack the thirst is extreme.

We quite agree with Holt that the most dangerous cases of acute gastro-enteric infection are those in which the bowels do not move. We saw such a case at Torresdale, with Dr. John Bacon. This baby was taken ill on a train coming from Pittsburg. For two days, his life hung in the balance; then with free catharsis, the evidences of his profound toxemia gradually subsided.

**Diagnosis.**—The diagnosis of acute gastro-enteric infection, should not be difficult, the history of the disease and the intensely acute onset resembling no other intestinal affection except Asiatic cholera. When the latter disease is epidemic, a bacteriologic examination is the only method of differential diagnosis. Occasionally it may be confounded with thermic fever (sunstroke), but the discharges of cholera infantum are continuous, and it lacks the high temperature which is always found in sunstroke.

**Prognosis.**—The prognosis of the disease is always grave. If, while the attack lasts, the child is fed on milk, the disease is nearly always fatal. The prognosis naturally is more favorable in strong, healthy children than in those who are weakly. A long-continued previous diet of artificial foods seems to make the prognosis less favorable; as does also the existence of rickets. The symptoms on which to base a favorable prognosis are a slight attack and rapid and steady decrease in the vomiting and diarrhea. The absence of symptoms of profound nervous depression are favorable. The physician, however, should not expect to save the lives of the majority of the infants affected with this disease.

**Treatment; Prophylaxis.**—The prophylactic treatment of acute milk infection consists in the careful attention to the cleanliness of the infant's food and the articles used in the nursing. If the child is fed from the breast, the nipples of the mother or nurse should be washed

before and after nursing according to the rules laid down in the chapter on hygiene and diet. If fed from the bottle, the greatest care should be exercised, particularly in summer, in scalding and thoroughly cleaning the nursing-bottle after each nursing. At all times, and especially during the summer months, any small digestive disturbance which may make its appearance must receive careful attention, and should a slight attack of diarrhea appear, milk must be stopped unless the child is nursing from the breast, and even then it is better to diminish the number of nursings and substitute definite quantities of barley water, etc. for several hours. Very often at this time a dram or two of castor oil, with small doses of calomel repeated once or twice during the twenty-four hours, will end the trouble. The city is no place for babies in the summer season. Country weeks, recreation piers, steam-boat rides, etc., may all help to save the city poor from this disease.

**Treatment of the Attack.**—The first indication in the treatment of milk infection—the one that is of the greatest importance—is to remove the source of the poison; hence all milk must be stopped, nor should any food containing it be given to the child. Sterilization or Pasteurization of milk does not render it a proper food in this disease. The child should receive no food at all for from 24–48 hours—indeed, in some instances as long as 72 hours—after the beginning of the attack. It is not starvation that causes the weakness and the eventual decease; it is toxemia. During this time small quantities of about half an ounce of cold sterilized water should be given at regular intervals, and in order to aid in stimulating the patient brandy or whisky may be added to the water. In some cases we have had favorable results from moderately large doses of alcoholic stimulants, giving say a teaspoonful of good brandy freely diluted in cold sterilized water every hour or even oftener during the period of collapse. The German method of giving tea at these times is also an excellent one. The second indication is to aid nature in freeing the system of the poison which is already in it. For this purpose washing out the stomach and intestines gives excellent results. These irrigations must be repeated frequently. The water must be sterilized and may be used plain, or, what is probably better, normal saline or Ringer's solution. The results of these irrigations are twofold: they not only aid in clearing the intestines of the poisonous materials, but also, by the absorption of at least a quantity of the solution injected, assist in keeping up the amount of water which the system needs and of which it has lost such large quantities by the continuous discharge from the bowels. In order to somewhat control the large watery movements, good results

sometimes follow the injection into the bowels of a pint of cool sterilized water containing from 15 to 30 grains of tannic or gallic acid. This injection should immediately follow a copious enema of sterilized salt solution. When the system has suffered a great loss of water, as in fact it always does in this disease, and a considerable degree of collapse makes its appearance, subcutaneous injections of salt solution are indicated. In order to maintain external bodily heat, the child should be placed in a hot bath, preferably a mustard bath, the temperature of which is to be increased from 95° F. (35° C.) to 110° F. (43.3° C.). In some cases placing the child in a hot pack at about 110° F. (43.3° C.) will cause reaction.

The stomach can best be irrigated with water containing .6 per cent. of sodium chlorid, after which from 3 to 5 grains of calomel may be similarly administered, as advised by Vaughn. The irrigation of the stomach and intestines should be repeated as long as the vomiting and purging continue. In the treatment of cholera infantum drugs must hold an inferior place; nevertheless, a few are useful. They may be administered by mouth or rectum, but the alkaloids used are best given hypodermatically. Taken all in all calomel is probably the best drug in the early treatment of this disease. From 1/2 grain to 1 grain is given in divided doses (1/10-1/20 of a grain). Such doses are given every fifteen minutes to every half hour. It is well to follow the calomel with some other laxative.

For the relief of vomiting, and also as a cardiac stimulant, hypodermic injections of 1/100 of a grain of morphin combined with 1/800 of a grain of atropin may be used. This is, indeed, a life saving measure. Digitalis or digitalin may also be used as a more permanent cardiac stimulant. As an intestinal antiseptic salicylate of soda has been recommended by A. Jacobi and Emmet Holt. The subnitrate or subgallate of bismuth, in doses of from 10 grains to a dram, the smaller doses to be administered by the mouth and the larger by the intestines, is certainly of some use. When the surface temperature is high—that is above 103° F. (39.4° C.)—an ice-cap may be placed on the child's head, or the fever may be reduced by frequently sponging the body with tepid water, followed by friction. No antipyretic drugs should be given, as they do more harm than good. When the surface temperature sinks below normal, stimulants in the form of a hot pack or hot bath should be employed, as has been before stated. Stimulating drugs, such as alcohol, aromatic spirits of ammonia, musk, camphor, or other agents of this class, may also be employed. After the vomiting has become

less frequent, the child may be given small quantities of nourishment in the forms described in acute and chronic enteritis.

Not until the child has passed at least a week without any return of the symptoms of the disease should milk be used as an article of food; it should then be carefully modified, either at home or, better, at some good milk laboratory. Great care must be taken as to the cleanliness of the food, the bottle, and the nipple. When the child has been fed by the breast at the time of taking the disease, the same rules must be adhered to as when it is fed by the bottle.

### SUBACUTE GASTRO-ENTERIC INFECTION

**Synonyms.**—SUBACUTE MILK INFECTION; SUMMER DIARRHEA; INFECTIOUS DIARRHEA; FERMENTAL DIARRHEA; FOLLICULAR DIARRHEA; SUMMER COMPLAINT

Subacute milk infection is a form of gastro-intestinal catarrh originating from the action of poisons generated by the growth and multiplication of bacteria in the milk from which the child is fed. The poisons are either bacterial or chemic, probably the former, and are either less intense than in acute gastro-enteric infection, or else they are operative in more resistant subjects. Probably because of more insidious beginnings, however, these attacks tend to persist for a much longer time; hence, the designation, subacute. The symptoms not being so severe as to cause alarm in the beginning of the disease, the patient is too often kept upon the same diet of infected milk, and thus continually receives a fresh supply of poisonous material. The number of deaths resulting from this disease is yearly much greater than from the acute form. It is almost entirely a disease of the summer months, and during this time its ravages are fearful. The number of its victims increases as soon as the temperature rises above 60° or 70° F. (15.6° or 21.1° C.), and decreases correspondingly with each cooler period. It is more prevalent in hot, damp weather than when the air is dry. Like the acute form, it is worse among the poorer classes of our great cities. Just in proportion as the milk given a child is pure and the patient's hygienic surroundings are good, just in so much is the danger of this disease decreased.

**Etiology.**—Subacute gastro-enteric infection, as has been before stated, is caused by bacteria, the poisons generated by which have usually been taken into the system in milk. The subacute form may also be but a simple continuation of a mild attack of acute infection for a period beyond the ordinary duration of the latter disease. We

have already called attention to the way in which these various types of diarrhea may merge into one another: Thus a baby with intestinal



FIG. 25.—SUBACUTE GASTRO-ENTERIC INFECTION.  
(De Witt's case.)

indigestion, is seized with an acute gastro-enteric infection. He recovers from the most acute symptoms, but continues to have a sub-acute diarrhea for some days, or even weeks.

**Pathology.**—Microscopic examination of the stools will show that they contain epithelial cells, crystalline formations, occasionally blood, and in older children fibers of meat. In one case recorded by J. Lewis Smith, he states that he observed particles resembling three or four crypts of Lieberkühn united and probably thrown off as the result of ulceration. The presence of bacteria and other toxins produce, first, an inflammation and secondly ulceration in the ileum and colon. These are minute follicular ulcers. (The “shaven-beard” appearance.) The greatest amount of inflammatory change is generally found in the colon, and here ulcers may be formed either singly or in groups. They may be seen in any part of this division of the large intestine. The upper parts of the duodenum and jejunum are generally free from these inflammatory changes.

**Symptoms.**—The disease usually begins with a gradually increasing diarrhea and some vomiting, both of which are increased after taking nourishment. The vomiting, however, does not always appear, and when it does, it is in the more acute form of the disease. Indeed, vomiting may prove fortuitous, serving to rid the little sufferer of much infected material. The symptoms may develop immediately after nursing, especially when the child is fed from the bottle, or may appear after an interval of cessation following an attack of acute milk infection. In this latter instance it almost invariably shows that the child has been returned too soon to a milk diet. As the bowel movements increase in frequency they will contain large quantities of mucus, undigested food, especially coagulated casein, and masses of fat. Each evacuation of the bowels is preceded and followed by the expulsion of gas. The color of the stools is at first yellow or brown, but soon changes to greenish or greenish yellow. Occasionally they are at first of a greenish color. The reaction of the stools is usually alkaline. They contain many bacteria, the colon bacillus or streptococci often predominating. Anorexia is present, and the child loses flesh and strength from failure of its nutritive powers. The tongue is coated with a whitish or grayish-white coat. The temperature is always increased during this disease, and its character is such that, except for the irregular time of the daily onset, it might be confounded with the fever of tuberculosis. The temperature is seldom above  $102^{\circ}$  or  $103^{\circ}$  F. ( $38.9^{\circ}$  or  $39.4^{\circ}$  C.), and may be so slight as to escape detection. During the attack the child is irritable, whining and crying in its sleep. After each bowel movement it generally enjoys a short period of rest. A considerable amount of flatulence is present, and this increases the amount of nervous disturbance. From the frequent irritating dis-

charge from the bowels the entire buttocks and often the greater part of the posterior surfaces of the thighs are covered with excoriations (intertrigo). Enlargement of the lymphatics of the groin, throat, or neck is not infrequent, and although this is not a serious complication, yet occasionally the glands may suppurate and cause trouble. Otitis media is an occasional complication. Various forms of irregular skin eruptions of an eczematous or erythematous character are often seen, and boils are a common complication. Aphthous stomatitis is a very frequent and painful accompaniment.

Bronchopneumonia and middle-ear disease from additional infections are not infrequent complications. Meningitis is rare, though hydrocephaloid is common enough. Sinus thrombosis (marantic) may occur. Death may occur from hypostatic congestion. Other diseases, such as nephritis or tuberculosis, may appear later in feeble children. Rilliet and Barthez first called attention to the frequency of nephritis in these cases, and Jacobi has championed their view. Recent studies, however, have demonstrated the infrequency of this occurrence (Morse). Edema, however, often amounting to anasarca is relatively common in the prolonged cases. It is probable that the toxins affect the permeability of the vessel walls. Spiegelberg has shown that most of the infectious complications result from other organisms than those invading the intestinal tract (mixed infections).

**Diagnosis.**—The diagnosis should be founded on the symptoms and history of the case. There are really very few diseases with which subacute milk infection can be confounded. From cholera infantum it is to be distinguished by the more gradual onset, its milder symptoms, and the absence of the characteristic large, watery stools found in the acute form of the disease; the vomiting and prostration are not so great, nor is the temperature so high as in cholera infantum. From the various forms of obstructions of the intestines it is to be diagnosticated by the fever, the gradual onset, and the absence of the extreme tenesmus, pain, and stercoraceous vomiting, none of which appears in subacute milk infection.

**Prognosis.**—The prognosis depends on the strength and vitality of the child, on its surroundings, and on the capabilities of those who attend its wants to give proper food and general care; the outlook for the recovery of feeble children—those who are fed on all varieties of foods and badly prepared milk and live amid unhygienic surroundings—is unfavorable. On the contrary, when the patient can be taken where good pure air can be breathed, especially the air of the seashore or mountains, and fed on properly prepared food, the prognosis is fairly

good. The duration of the attack has also a considerable influence on the prognosis. The sooner the patient has the care of a physician, the better the prognosis.

**Treatment.**—As in all other forms of intestinal disease caused by the presence of bacteria and their toxins, the careful regulation of the diet is the most important consideration. So long as milk, which is one of the best of all culture-media for bacteria, is given the child, just so long will the disease continue. The removal of this article of diet is as much demanded in subacute infection as it is in the acute form. These children, then, should be relieved entirely from a milk diet, and placed upon substitute feeding. The plan already outlined (see infant feeding) is usually a safe one: A day of starvation, with water in abundance; as second day of barley water, with or without stimulants; a third day of sweetened barley water; the fourth, fifth, sixth and seventh days with equal parts of albumin water and barley water, sweetened with lactose, maltose or cane sugar (saccharose). Finkelstein's method (albumin milk) may be tried in the very weak cases. The child must not be placed on a milk diet until the disease is well under control, the discharges from the bowels have become normal in color and frequency, and vomiting has ceased. Buttermilk is often better than milk even at this period. The patient, if living in a crowded tenement, should, if possible, be taken to the country, or at least where it can breathe pure air. Directions should be given the mother or nurse not to hold it in the arms any more than is necessary, but to let it lie on a moderately hard mattress where it can have the air from an open window, or, better, on a hard pillow in its coach in a cool place in the yard, or on the street after the sun goes down. Instruction should be given not to allow the child to have the nursing-bottle at its lips all the time while lying in the coach or on the bed, but it should be fed only at regular intervals, and between these should receive nothing in the way of nourishment. It should have at least one bath daily, and the diapers should be thoroughly washed and boiled. It is our custom to make the nurse or other attendant place all of the soiled diapers in a covered jar. Then the physician may view the whole fecal field. His study completed, an antiseptic solution may be poured right into the jar. They should be changed frequently.

Irrigation of the intestines is of as much importance in the treatment of this disease as in other forms of infective diarrhea. Lavage of the stomach is indicated when the mucous membrane of this organ seems to be involved. When the patient is seen at the beginning of the attack, the greatest benefit can be derived from the giving of a laxative,

such as moderate doses of calomel, followed by castor oil. When we see the patient later in the disease castor oil and rhubarb are the only safe laxatives to use. Both tend to leave constipation in their wakes. The various salts of bismuth and other intestinal antiseptics are of great use. A splendid combination is:

R. Bismuth salicylat.....	gr. xxiv
Bismuth subnitrat.....	℥ii-℥iij
Mist. cretæ.....	fl. ℥j
Aq. cinnamon.....	q.s. ad. ft. fl. ℥iij
M. et solve.	
Sig.—fl. ℥j every two or three hours. (For an infant one year old.)	

Opium should never be employed until the fever has been absent for some days and the stools have lost much of their odor. Then, it may prove a useful drug to slow peristalsis. After the disease is thoroughly under control and the digestion remains weak, such remedies as nux vomica, arsenic, particularly in the form of Fowler's solution, nitromuriatic acid, and others of this class may be used with benefit. Iron may be employed if anemia is present. Cod-liver oil has been recommended, and is undoubtedly of use, but when the digestion is weak, as it generally is, it is best employed in the form of inunctions.

## ILEOCOLITIS

Ileocolitis, the so-called dysentery or dysenteric diarrhea, is an inflammation of the mucous membrane of the lower part of the large intestine (colon and rectum), accompanied usually by the formation of patches of ulceration, which in number may be single or multiple. The disease may be acute or chronic, and while usually sporadic, sometimes appears in an epidemic form.

**Etiology.**—The same causes which in children produce other forms of intestinal inflammation are also active in the etiology of dysentery. Improper feeding with bad milk, improper hygiene, crowding in poorly ventilated, filthy tenements, and general lack of care are all fruitful factors in the causation. The disease is more frequent among the poorer classes, but it will occur in any condition of life where children are not given the proper kinds of food. As a rule, the disease is more common in cities and in the "slums" of cities than in the healthy districts of the latter or in the country. Predisposing factors in the etiology of the disease are all lowered states of vitality, such as those produced by rickets, syphilis, tuberculosis, etc. The dis-

ease is probably always bacterial in origin, though the only organism that has been shown to bear anything like a constant relationship to this disease is the bacillus dysenteriae (usually the Flexner-Harris type). Duval and Bassett, two medical students, demonstrated this in 1902, and, in the following year, the investigation of the Rockefeller Institute verified their results. In 412 cases studied, the bacilli were found in 270. Unless studies are pursued very early in the disease, however, pyogenococci are also found in the stools. In a recent case, seen by one of us, only staphylococci were found. Unquestionably these pus organisms play important rôles clinically as they do in so many of the infectious diseases. The path of infection in the majority of cases is probably through the food or drinking-water. The disease occasionally appears in epidemics, the cause of which is decidedly obscure, unless, like typhoid, it be carried by water, or air infected from a previously existing case.

The croupous or diphtheric form may result from diphtheria in any part of the air-passages, or it may appear as a primary infection of the entire colon or lower part of the ileum or cecum. A rare disease, particularly in childhood, known as amebic dysentery, has been described by Osler, Holt, Councilman, and others. It is caused by the amœba coli (Lösch) or ameba dysenteriae (Councilman and Lafleur). Osler describes the ameba as follows: "It is a unicellular, protoplasmic, motile organism from ten to twenty micromillimeters in diameter, and consists of a clear outer zone (ectosarc) and a granular inner zone (endosarc) containing a nucleus and one or more vacuoles." This disease is seldom seen in this country, but it is very frequently in the tropics. The source of infection is probably drinking-water.

**Pathology.**—We find the mucous membrane of the colon and rectum congested to a very marked degree, or occasionally the inflammatory condition may extend upward as far as the ileum, or in bad cases the whole large intestine may be involved. The mucous membrane is intensely hyperemic, and this condition may be limited to circumscribed areas or may be general. Small hemorrhages sometimes take place into the mucosa or submucosa. The mucous membrane in the parts affected is covered with a thick, sticky mucus, which in some cases is extremely adhesive and hard to remove. The color of the mucous membrane varies from bright red to purple, and is seldom uniform. The solitary lymph-follicles along the colon are swollen, and each is surrounded by an area of hyperemia, and in many cases the breaking-down of these glands occurs later, producing an ulcer. These ulcers may be either single or multiple, and often extend considerably in size

after they first begin, or in some instances two or more may coalesce, forming one large, irregular, ulcerative patch, which may simply be confined to the mucous membrane, or, proceeding deeper, may involve the submucosa or even perforate the entire wall of the intestine. The mesenteric glands are enlarged and softened. The liver is usually congested, although anemia of this organ may be present, as in the case recorded by Busey. Suppuration of the kidney may also occur. Examination of the brain will not infrequently show one or more thrombi in the sinuses of the dura mater. In other cases inflammation of the brain structure or, on the contrary, cerebral anemia may be found.

Croupous or diphtheric dysentery is the name given to a variety of the disease which is associated with the formation of diphtheric ulcers occurring in the same part of the intestine as do those of the catarrhal form. It is not always caused by the Klebs-Loeffler bacillus. These patches are covered with a tenacious, grayish-white membrane which, when removed, leaves a bleeding ulcer. The membrane is composed of fibrin, necrotic cells, and blood-corpuscles. Both the mucosa and submucosa undergo considerable infiltration and thickening. Between the patches the mucous membrane is congested and more or less roughened. The pathology of the amebic form is as follows:

The lesions are principally found in the lower portion of the ileum and colon, and consist of small elevations appearing along the mucous membrane and associated with infiltration. The ulcer first begins as a small papule, the upper part of which sloughs off, leaving a grayish-yellow ulcerating surface. Amebæ are found in the tissues in and around the ulcers, in the lymphatic spaces, and occasionally in the blood-vessels. Multiple abscesses arising from the same cause are usually found throughout the liver and occasionally in the lungs.

**Symptoms.**—In the so-called acute catarrhal form of ileocolitis the attack begins suddenly with diarrhea, accompanied by great tenesmus and followed by chills and a moderate rise of temperature. As the disease progresses the patient rapidly loses strength, the pulse becomes rapid and feeble, and the face presents a peculiar pinched, pale, and anxious expression. The weakness is increased after each evacuation of the bowels, the number of passages soon becoming very numerous. The stools at first contain ordinary fecal matter, but rapidly become smaller in quantity, more liquid, and mixed with mucus, blood, or pus, and in advanced cases contains shreds which are sometimes described as resembling the washings of raw meat. This symptom is generally associated with considerable ulceration. The

urine is scanty and high colored, and in bad cases there may be absolute suppression. Vesical tenesmus is a common symptom. The abdomen is usually swollen and tympanitic. The tongue is covered with a brown fur along the center, its margin being red. Vomiting may occur, but is more generally seen in the earlier stages of the disease; it is usually not severe. If the case progresses toward a fatal issue, the respirations become irregular and sighing, the eyes are partially closed, and the pupils are dilated. Not infrequently the child will die in a state of absolute collapse. Prolapse of the rectum frequently occurs, and is caused by the great state of relaxation in this part of the bowels. In the later stages of the disease convulsions are common, and the child may die during an attack. Microscopic examination of the stool shows large quantities of mucus, with epithelial cells of different types usually found in the lower bowel, blood-corpuscles and pus-corpuscles, fat, and a large number of bacteria. In the so-called diphtheric form of the disease the symptoms are about the same as before described, except that they are more intense. The amebic form is characterized by a series of symptoms not unlike those of the catarrhal and diphtheric varieties, though less severe than the latter. Large numbers of the *amœba coli* will be found in the stools during the diarrheal attack and will serve to verify the diagnosis.

**Diagnosis.**—The diagnosis of acute cases of the catarrhal form must be made by the character of the stools and by the general symptoms. The following are a few of the other types of intestinal catarrh from which it must be differentiated: From acute milk infection it is to be differentiated by its less acute onset, less amount of vomiting, and higher temperature. In dysentery the stools are smaller in quantity, contain blood, mucus, and pus, and have less odor. In ileocolitis there are not the general symptoms of acute poisoning which are always present in milk infection. From other forms of gastric or enteric catarrh dysentery can be diagnosticated by its smaller stools, intense tenesmus, and the amount of prostration following each evacuation. The stools of dysentery are distinctly bloody, and there are less of mucus and water than in the stools of other forms of enteric catarrh. In dysentery the stools lack the greenish or greenish-yellow color of those seen in other forms of inflammation affecting the upper parts of the intestines. The diphtheric form is seldom seen in infants, and, indeed, is very rare, even in older children. Its onset may be either very rapid or slow, but the symptoms are much more severe than those of the acute catarrhal variety. The pseudomembrane found in the stools greatly aids in the diagnosis. Amebic dysen-

tery is a rare disease in this climate. When found, the stools will be seen to have a grayish-yellow color and contain blood and mucus (Adams). Its diagnostic points are the recurring attacks of diarrhea and the presence of amebæ in the stools.

**Prognosis.**—The average duration of the acute attack of catarrhal ileocolitis is from a week to ten days, and with proper treatment should end favorably. The prognosis varies, however, when the disease is epidemic, the mortality in some epidemics being very high. Even the acute variety may end fatally in from twelve to thirty-six hours. Favorable indications are bowel movements of moderate amount and decreasing frequency, small quantities of blood being passed, with slight tenesmus and not a great deal of weakness following. A good, strong heart action, the absence of nervous depression, and no convulsions are all evidences of a light attack of the disease.

**Treatment.**—The treatment of ileocolitis may be divided into preventive and curative. The former may be secured by a careful oversight of the child's diet and hygiene, the same rules being followed as have been given in the treatment of other forms of intestinal disease. These rules should be carried out with especial strictness and care during the summer months, extra precautions being taken when the disease is epidemic, remembering especially that no attack of indigestion in a child, however slight, is too small a thing to warrant the attention of the physician. Many a fatal attack of dysentery could be checked in the incipient stage were intelligent medical aid called at this time. Treatment should be initiated with broken doses of calomel, and a full dose of castor oil should follow the mild chlorid. Milk in all forms, even breast milk, should be immediately stopped in the presence of this disease. Starvation for at least a day should be considered as an imperative procedure. Water, however, may be given freely, provided that it is sterile. Carbonated water is useful where there is much vomiting. The first foods that should be given are those recommended in acute and subacute milk infections; viz., the carbohydrates. Barley water we give the second day, and dextrinized barley jelly on the third. Milk is never given under a week's time, and then it is always prescribed in a weak predigested formula, or in the form of buttermilk. Kerley has unquestionably proved Jacobi's contention that the carbohydrates are the safest food substitutes in the gastro-intestinal disorders. As disturbers we would place the following foods containing proteids in this order: 1. Milk; 2. whey; 3. beef-juice; 4. albumin water. In the more chronic forms of ileocolitis, one may have to employ (cautiously) one of the

three last mentioned; but if disturbance follows, that food should be immediately withdrawn. If possible, remove the child to the mountains or seashore, the latter frequently proving the better. In both, however, it is hard for the physician to so carefully supervise the diet of the child, for this must receive the greatest care even after the symptoms of the acute attack have ceased. Daily sponge-baths should be given all through the attack. If the patient is an infant, a very important point is the disinfection of the diapers; these should be well boiled and soaked in a solution of corrosive sublimate, carbolic acid, or chlorin preparations as soon as the attending physician has examined them. It is still better to substitute non-absorbent cotton and gauze for the ordinary diaper.

In dysentery, as in other forms of intestinal inflammation, the greatest benefit is always to be derived from local treatment by intestinal irrigations; here it is of importance that the irrigation, while it should be copious enough to thoroughly flush and cleanse the bowel, should be allowed sufficient means of ingress and egress. It is better that the bowel should not be distended to an extent sufficient to produce pain; and it should also be remembered that the lining of the large intestine is thinned by ulceration, so that there is always at least a moderate danger of perforation when the liquid is forcibly injected or the bowel is overdistended by too much fluid. Irrigation can best be accomplished by a soft-rubber catheter. This should be well oiled and introduced with very great gentleness to a distance of three or four inches. A quart of water may be used in each irrigation in a child from two to five years old. The return flow must be watched to see that the fluid is not retained. The gravity or bag syringe is the only safe form to use for this purpose, and the reservoir should not be over one foot above the patient's buttocks. A safe way to prevent too much pressure is to insert the catheter over the small end of a medicine dropper, and the rubber tube of the fountain syringe over the other. The water used in the irrigation may be medicated with any one of the numerous intestinal antiseptics, but in many cases warm sterilized water to which one dram of sodium chlorid to the pint of water will give good results. Many authors claim to have excellent results by using irrigations of iced water, or by gradually lowering the temperature of the water during irrigation. This, we believe, should never be done unless there is high temperature. The number of irrigations must depend on the amount of diarrhea present; as this lessens, they may be given at increasing intervals. As a rule, they should not be given more than twice a day, and as-

tringent injections not more than once daily. Of the latter, tannic acid (gr. xv–gr. xxx to one pint of water) as recommended by Holt, and silver nitrate solutions (1–3000 to 1–2000) may prove of vast value. In a recent case the latter solution acted well nigh miraculously. When the pain and tenesmus are increased greatly by the introduction of the tube, the rectum may be anesthetized by cocain in 1 per cent. solution, the agent being used alone or with carbolic acid; such solutions may be brushed over the mucous membrane or the drugs introduced in the form of a suppository with cocoa-butter. Some authors contend that the same effect is produced by a small ice-bag applied to the anus or by an ice suppository.

Out of all the large number of drugs, antiseptic and astringent, which have at various times been recommended as cures for this disease, there is no one upon which we can absolutely rely as a specific. Complex prescriptions should be avoided, and the innumerable new remedies should be tried with very great caution, as through their use in many cases the physician does more harm than good. Of the astringent antiseptics, the best are probably bismuth and its salts, particularly the subgallate and subnitrate.

The mineral astringents give their best action after the disease has progressed for some little time, and it is then that the bismuth salts and a few of the intestinal antiseptics, particularly salol, give their best results. Tannigen and tannalbin sometimes act very well, but they should not be used until evidences of infection have subsided. For the pain and tenesmus, analgesic suppositories of the formula given below are useful, one every hour or more:

R Cocain hydrochlorate.....	1 grain
Aqueous extract of ergot.....	12 grains
Aristol.....	5 grains.
Cocoa-butter, to make twelve suppositories.	

Note: Cocain should be watched very carefully. It is a dangerous drug in young children.

Preparations of opium should be used with extreme caution in dysentery in children. When the disease has progressed for several days, the camphorated tincture may be used with safety, and may be combined in small doses with any of the before-mentioned drugs. Naphthol, the sulphocarbolate of zinc, and bichlorid of mercury have all been used and have their advocates. Alcoholic stimulants are especially indicated when weakness is great, and hot fomentations may be applied over the abdomen. Enemata of small quantities of opium and starch water—five or six ounces of warm mucilage of starch and

boric acid, with ten minims of tincture of opium, will often be of service after the disease has continued some days.

In amebic dysentery Councilman and Lafleur recommended the use of solutions of quinin, in a strength of 1:5000 to 1:1000, by intestinal irrigation. Injections are given with the patient in a knee-chest position.

Recent reports seem to indicate that highly beneficial results have been obtained in the treatment of amebic dysentery in adults by the use of hypodermic injections of emetin, an alkaloid of ipecacuanha. Favorable reports on this subject have been given by Rogers,<sup>1</sup> Vedder<sup>2</sup> and others. It would seem probably that this same treatment might give as good results in the same disease in children.

The treatment of diphtheric dysentery is practically the same as in other forms.

#### AMYLOID DEGENERATION OF THE INTESTINES

This form of intestinal disease is most commonly found associated with the same changes in other organs, particularly the liver, spleen, and kidneys, where it usually appears as the result of prolonged suppuration. More rarely it is associated with syphilis. The ileum is most apt to be attacked. The disease is rare in infants, but is occasionally seen in older children.

**Pathology.**—The changes begin in the walls of the capillaries and small arteries of the intestinal villi, but later the submucosa, and even the mucous membrane of the intestine, may be affected. The latter has the characteristic pale, semitranslucent appearance which is found in amyloid degeneration elsewhere. The same chemic tests as are used in the recognition of amyloid diseases of the liver are of service here. Amyloid degeneration of the intestines has no special symptoms.

The **treatment** is entirely symptomatic.

#### CHRONIC CONSTIPATION

By chronic constipation we understand a condition in which the contents of the bowels are evacuated with less than normal frequency and in less than normal amounts. The consistency of the evacuations is also increased, so that the effort of emptying the bowel is attended with considerable pain and muscular effort. Thus constipation implies a lessened number of stools and an increased consistency of the same. During the first year of life an infant may have from two to four, or even

<sup>1</sup> Dysenteries, their differentiation and treatment, by Leonard Rodgers, M. D., F. R. C. P., B. S., F. R. C. S., etc., London. Henry Frowde, New York, Oxford University Press.

<sup>2</sup> Origin and present status of the emetin treatment of amebic dysentery, by Edward P. B. Vedder, M. D., Captain Medical Corps, U. S. A. Journal of the American Med. Asso., Feb. 14, 1914.

five, discharges without being considered to have had more than the normal number. This is not usual, however; most babies have but two stools after the first few weeks of life. Indeed, if a single daily stool has a normal consistency, the baby is not said to be constipated. In the second year of life the number of evacuations is generally about two or three a day, and from that time on they will diminish until the individual has one normal stool a day. Constipation may be described under two forms: the atonic and the spasmodic. The atonic form is generally due to a lack of the proper peristaltic motion of the intestines. In the spasmodic form the fecal movements are usually increased in size, and are much harder than normal; this will frequently produce an irritable condition of the rectum, so that the pain of a bowel movement is so great that the child will not willingly endeavor to have a fecal evacuation. Anal fissures may add to the child's suffering and his fear of effort at stool.

**Etiology.**—The causes of constipation are varied; thus, the conformation of the large intestines in the child may in itself be a cause, as during this period of life the ascending and the transverse colon are shorter, and the descending colon is longer, than in the adult. In the child there are a greater number of curves of the convoluted sigmoid flexure; the culdesac of the sigmoid is deeper, this being especially noticeable just above the rectum where actual angulation may be observed. The small space in the interior of the child's pelvis into which many abdominal organs are crowded may tend to produce constipation. The imperfectly developed condition of the intestines themselves and their lack of general muscular tone are both factors in the production of flexions or twists, especially in the lower bowel. It is also claimed that there may be persistence of the rectal valves. Again, the levator ani muscle does not play the efficient rôle that it does in later life. Last among the mechanical causes is an undue spasm of the sphincter ani muscle. One of us has seen permanent relief follow a single stretching of the sphincter. Food, also, is a very important causative factor, especially when this is in a state of partial fermentation. Milk itself, usually the proper food for the child, will, when in a condition unadapted to the digestive organs, produce constipation, and a continued diet of cereals is apt to give the same results. Rickets is a potent cause of muscular atony as Jacobi contends, and we agree with him that constipation resulting from the atony of unstriated muscular tissue is often the first clinical evidence of rickets. A deficient excretion of bile may also be a cause. Various malformations, etc., may act as etiologic factors. Inflammatory causes, particularly appendicitis, are all too potent. The pressure of tumors on the bowel may also produce

it. Constipation usually follows the administration of certain laxative drugs after the period of their action has ceased.

In the majority of mild cases no pathologic changes are noticed. When the condition has been intense and has lasted for considerable time, there is frequently some irritation of the lower bowel, accompanied by a catarrhal condition, giving rise to symptoms that may for a time simulate diarrhea. In bad cases an actual ulceration of the bowel may result, although this is rare.

**Symptoms.**—The symptoms other than the constipation itself are, for a time, more or less obscure. However, there are generally present some headache, restlessness, occasionally a slight rise of temperature, distention and tenderness of the abdomen, and upon examination the transverse colon can generally be outlined by palpation and percussion. Scybalous masses are often detected. In very severe cases there may be dyspnea, and even convulsions of eclamptic type. Palpitation of the heart and vertigo are often present. If the condition continues for some time, the child loses its appetite, becomes restless and fretful, complaining of almost constant abdominal pain, generally expressed by crying and drawing up of the legs.

**Diagnosis.**—A careful elicitation of the history and an examination of the stools cannot fail to make the diagnosis. The etiologic diagnosis is another matter and requires a searching inquiry, by one acquainted with all the possible causative factors. It is particularly incumbent upon the diagnostician to exclude inflammatory possibilities before adopting a treatment. The skilful roentgenologist can help us to determine mechanical causes, etc.

**Prognosis.**—This too depends upon a knowledge of cause. We agree with Wm. Cheadle, that constipation may always be cured in infancy, childhood and adolescence, provided that there be no congenital anomaly of the gastro-intestinal tract to account for it.

**Treatment.**—We are dealing with a chronic condition, and to the student that should always mean, *that he investigate and regulate every hygienic detail of the patient's life*. Again, successful treatment must necessarily depend upon the age of the patient and the cause of the disorder (See etiology.) In young babies the food is often at fault, and in most cases dependent upon dietetic factors, the fat is deficient and oft-times the proteid in actual or relative excess. Regulation of the mother's life in the case of the suckling and carefully devised formulæ with the bottle-fed may bring us success. Nevertheless, it is a false assumption that an increase of the fat element will always cure infantile constipation and much harm may result from following out such practice. Far more commonly, in early infancy, the mechanical factors (chiefly

in the lower bowel) are at fault. If the mere insertion of a gluten suppository produces the desired result, then that seems the most rational treatment. Indeed, this measure may be put to a most useful purpose, for it enables the mother to slip a chamber under the infant's buttocks after using the suppository. This done at the same time every day trains the infant in regular habits. The same thing may be said of small enemata, though they should be abandoned if larger and larger amounts are required daily. One of us is very fond of the following injection: One and a half ounces of warm soapy water; 1 to 2 drams of olive oil and 1 dram of the milk of asafetidæ. Abdominal massage, properly performed, is also a valuable adjunct in treatment at this period of life (see below). Rickets should receive its proper treatment when it is obviously the underlying cause.

Cheadle, of London, has advanced most forceful and lucid views on this subject—views that we can most heartily endorse. In treating infants during the second year of life and in dealing with children, we keep them ever before us. Substantially they are these: 1. Our first clear indication is to keep the intestinal contents more fluid. 2. The unstriped muscular tissue of the intestinal wall, being in a state of atony, our second indication is to stimulate peristalsis (*i.e.*, to make the muscle exercise), not at irregular and infrequent intervals, but frequently and with regularity. It is refreshing in these days of therapeutic nihilism to find that he believes chronic constipation may be cured largely through the use of drugs. How then may these clear indications be met?

1. Keeping the intestinal contents liquid:

This may be achieved:

A. By using proper quantities of water. Prescribe water as accurately as drugs are prescribed. The glass of water, very hot or very cold, (Weir Mitchell) before breakfast, may serve the additional purpose of exciting peristalsis.

B. But the mere imbibition of water is not sufficient, as it is rapidly absorbed from the large bowel. Drugs that produce watery stools (the salines and podophyllin) are therefore indicated. Cheadle gives these in small amounts and at regular intervals, often three times daily.

2. The stimulation of peristalsis:

C. Most efficient for this purpose are the vegetable laxatives, particularly cascara, senna and rhubarb. These too should be used frequently and regularly, in small though sufficient dosage.

D. The diet is, of course, important; but we agree with Cheadle that results should not be produced by mere irritation, as they are too

often in the use of figs, dates, etc. If intestinal catarrh results, we tend to defeat our purpose. A careful dietary should be laid out for the individual case, and in it the sub-acid fruits (oranges and grape-fruits), stewed fruits, green vegetables and cream, should occupy prominent places. Fruits with seeds, particularly berries, are contraindicated.

E. Mechanical measures—massage, hydrotherapy, and electricity: These, the so-called natural methods, are useful adjuvants. Massage should be performed by a skilful person or a masseur should instruct the mother or nurse. Begin with rotary movements around the navel. Then follow the course of the large bowel with stroking movements. Do not pursue these movements for more than three minutes at first, though later, the time may be increased to five or ten minutes. In older children, grasping movements of the colon may follow the strokings. Have a care, however, that no inflammation exists. In adolescents, cannon-ball massage may act mechanically though in general, we should not advocate it. Active movements (as in the Delsarte mattress work) are often useful, serving the double purpose of strengthening the abdominal muscles and increasing the local blood supply. Hydrotherapy we often employ, either in the form of the cold sponge over the abdomen, that follows the tepid bath, or in alternate douchings with hot and cold water over the same region. Electricity, usually the interrupted faradic current, has acted well in some cases. In older children, one electrode may be placed in the rectum.

Useful prescriptions are:

R. Sodii phosphat..... ʒi-ʒij  
 Ext. senna fld..... fl. ʒss-fl. ʒj  
 Glycerin..... fl. ʒij  
 Aq. cinnamon ..... q.s. ad. fl ʒiij  
 M. et solve.

SIG.—fl. ʒj three times daily, before eating.

or

R. Spt. chloroform..... ℥xxiv  
 Sodii sulphat..... ʒii-ʒss  
 Ext. cascar. sagrad. fld..... fl. ʒii-fl. ʒss  
 Syrup. lactucarii..... q.s. ad. ft. fl. ʒiij  
 M. et solve.

SIG.—fl. ʒj three times daily, before eating.

or

R. Resin. podophyll..... gr. j  
 Aloin..... gr. i-iiij (according to age).  
 Ext. cascar. sagrad..... gr. xii-gr. xxiv  
 Mel q.s. ad. ft. confection.  
 Div. in confection. no xii.

SIG.—One confection at bedtime.

## INTESTINAL COLIC

The term intestinal colic is usually applied to attacks of severe griping pain, paroxysmal in character, occurring in the intestines, and unaccompanied by inflammation. It is to be distinguished from the colic of appendicitis, intussusception, gall-stones, strangulated hernia, and lead or arsenical poisoning.

**Etiology.**—Intestinal colic is really a symptom and not a disease. It is most frequent in the first year of life, and is probably the most common form of pain during infancy. As seen in infants and young children it is usually due to flatulence produced by a mass of undigested food, chiefly milk. In infants up to six months of age its most frequent cause is a milk too rich in proteid or one in which the casein is in large masses or so tough that the digestive secretions cannot decompose it. A milk overrich in fat, and much more rarely sugar, may cause colic. The farinaceous foods long continued in young infants very frequently produce intestinal distress. It is considerably more frequent in bottle-fed babies than in those fed from the breast, although it is seen when the mother's milk has become disturbed by continued bad food, excitement or worry, taking cold, or disease, or when it may be too rich in proteids. Colic is frequently seen when the maternal milk-supply is limited in quantity or when the colostrum milk has lasted beyond the usual period, and may continue while the mother is in bed. A very common cause is frequent and irregular feeding, whether by breast or bottle. Pregnancy or menstruation in the mother may cause her milk to give the child colic. On the part of the infant, a naturally enfeebled digestive tract or general nervous system is a predisposing cause, as is also dentition.

In older children colic is frequently caused by errors in diet, fruit-seeds, foreign bodies, intestinal parasites, and rarely plumbism. It not infrequently follows wetting the feet or exposure to cold and damp. As has been before stated, colic is a symptom of appendicitis, gall-stones, strangulated hernia, and other forms of intestinal inflammation, and these must always be borne in mind in studying a case.

Intestinal colic is usually associated with flatulence, the latter being caused by gas produced by decomposing food or secretions distending the intestines and which cannot be expelled, but there is in many cases associated with this a spasm of the muscular coats of the intestines which is chiefly reflex. This latter is the chief cause of the pain. In some cases there is no distention of the intestines, but only

reflex spasm of their muscular walls; these latter cases are mostly the result of taking cold or getting the feet wet.

**Symptoms and Diagnosis.**—The symptoms of intestinal colic are so well known as to make a lengthy description unnecessary. When the attack is sudden, the child's features, which have been placid a moment before, become contracted, the face blanches, and in bad cases the fontanel always becomes depressed. The lower extremities are drawn upon the abdomen, the arms are flexed, and frequently the thumbs are also flexed and adducted. In male children there is contraction of the scrotum. The cry is loud and paroxysmal, and is expressive of sharp acute pain. The abdomen will be found hard, tense, and somewhat distended. The attack may be followed by expulsion of gas, and when this occurs, the symptoms quickly subside and the child falls asleep, probably to be awakened in a short time by another attack. The intervals between the paroxysms may be considerable, or the attacks follow one another in rapid succession and seem continuous. The symptoms may be slight in degree or severe enough to cause severe prostration, the child being covered with perspiration. During the attack the pulse is somewhat accelerated, and the temperature in severe cases may be a little raised. Not infrequently the symptoms subside after taking food, particularly if it is warm. In many infants who are habitually badly fed, the colic is almost constant, and in these it is often hard to distinguish between the cry of hunger and that of colic; in both cases food is taken eagerly and is generally followed by temporary relief. The cry of colic, however, is more violent and paroxysmal than that of hunger, and as the pain quickly returns after taking food, the child will again cry more violently than before. In colic there are also the other symptoms expressive of pain. The cry of hunger is more continuous and not so shrill, and feeding is quickly followed by continuous relief, the child falling asleep. There are no symptoms of general pain. The cry of colic in infants several weeks old must also be distinguished from the cry of habit. The abdominal pain of appendicitis, intussusception, or hernia must be distinguished by the diagnostic symptoms of each condition (see articles on these subjects). An accompanying gastric colic is sometimes seen.

**Treatment.**—The treatment of an attack of simple colic consists in helping the intestines to unload themselves of the gas and decomposing material causing it, and to relieve the pain. For the first two there is nothing better than an enema of a half pint of luke-warm water, or, if this fails, Holt advises a second irrigation of cold water in which a teaspoonful of glycerin is dissolved. The enema may be

made more efficacious by the addition of a few drops of spirits of turpentine or one fluidram of milk of asafetida. Dry heat may be applied externally by means of cloths or a hot sand, salt, or water-bag. The child's feet should be kept warm by a hot-water bottle or bag. There are a few drugs which, administered internally, may do some good. Frequently a full dose of castor oil will relieve in a short time. Calomel in small doses repeated will often do good, or, one grain, with bicarbonate of soda, two or three grains, given before each nursing will sometimes give relief. Probably the quickest effects are to be obtained from Hoffmann's anodyne or paregoric, given in doses of from two to five drops. The use of preparations of opium, however, is not advised, as they do more harm than good.

Louis Starr and Hare recommend the following:

- R. Chloral hydrate ..... gr. xvj  
 Potass. bromid. .... gr. xxxij  
 Aq. menth. piperit. .... fl ʒij.
- M. SIG.—A teaspoonful in a little warm water every four hours for a child six months old.

Asafetida, (particularly in an enema.—See last chapter), peppermint, soda-mint, the bromids, and arsenic are among the remedies most commonly employed. Between the attacks the diet should be regulated, and when this is properly done, the colic usually disappears of itself. This attention to the diet must be directed toward the composition of the food. When the proteids are high in the breast milk, one may tentatively give warm or tepid water before each nursing; while at the same time he adopts the methods Rotch has suggested, of cutting down these constituents of the mother's milk by diet and exercise. If water fails as a diluent we advise the use of two drams of half strength barley water before the nursings; or, this failing, of a dram each of barley water and lime water. When the infant is fed artificially, the proteids should be lessened in the milk mixture given until a formula is prescribed which can be digested without pain. In the weak baby or in one whose digestive functions are feeble the use of the hot-water bag in the crib serves to avert many an attack of nocturnal pain.

### Intestinal Obstruction

Under this title will be described together those forms of partial or complete occlusion of the intestinal canal, either of sudden or gradual onset, which are most commonly found in children. Among these we may include: (1) Strictures arising from organized plastic

bands or adhesions, the result of abdominal inflammation by tumors or ulcerations; (2) strangulations due to various forms of hernia; (3) intussusception or invagination of the bowel; (4) volvulus, or twisting of the bowel upon itself; (5) mechanical obstructions caused by masses of feces or foreign bodies. The last has been already dwelt upon at some length in the article on Chronic Constipation.

### INTUSSUSCEPTION

**Intussusception.**—In children the most common form of obstruction is that known as intussusception or invagination of the bowel. In the majority of cases the invagination is direct and consists in a slipping of one segment of the bowel within the other (like a glove-finger inverted on itself), the invaginated part being in nearly every case the portion which is furthest away from the anus.

**Definitions.**—In a certain number of cases the order of the invagination is reversed, the lower segment becoming invaginated into the upper. This is known as retrograde intussusception. The original form, which has been mentioned, is known as direct intussusception. Intussusception involves three layers of bowel, each layer consisting of all the intestinal coats. The outer layer is known as the intussusciens, sheath, or receiving layer, while the internal is known as the entering layer, which, together with the middle or returning layer, constitutes the invaginated part or intussusceptum. The junction of the middle and inner layer is known as the apex. Intussusception may be double or even triple. In the former case five layers of intestines are involved, while in the latter seven layers have been found. While invagination of the bowel may occur in almost any portion, it is stated that one-half of all cases occur at the junction of the small and large intestines. When in this position the ileum becomes invaginated into the colon, the condition is known as ileo-colic intussusception. In less than one-third of the whole number of cases invagination takes place only in the small intestine. This is described as ileal or jejunal intussusception. In a small number of cases the invagination may occur only in the large intestine. This is known as colic intussusception. In most cases invagination begins in the right side of the abdomen, but in all the varieties except the first the position of the neck continually changes, owing to the following reason: As the entering layer, or intussusceptum, passes into the sheath, it carries with it a certain amount of mesentery, causing a considerable degree of traction, which produces a curving or bending of the intussusceptum toward the mesenteric side of the receiving portion. In

the most common variety, the ileocecal, the invagination begins on the right side of the abdomen, but as the increase is particularly at the expense of the large intestine, the tumor will, by the time it has grown large enough to be felt by palpation, be found on the left side. It is quite possible that in some cases the ileocecal valve, with the apex of the intussusceptum, may protrude from the anus, and occasionally the apex may be detected by digital exploration of the rectum when actual extrusion does not take place.

**Etiology.** *Predisposing Causes.*—It is a well-assured fact that intussusception occurs more frequently in infants and children than in adults, and in males oftener than in females. It has been stated (“American Text-book of Surgery”) that more than 50 per cent. of patients are under ten years of age. It is without doubt the most common form of intestinal obstruction in children. Heubner states that three-fourths of all cases of obstruction of the bowel occurring in childhood are from intussusception. The probable reason for this may be that the descending colon during infancy is of relatively greater length than in the adult, while the mesocolon is wider, thus making easier a displacement of the former.

*Exciting Causes.*—The exciting causes of intussusception in many cases are obscure. According to some authorities, a portion of the bowel may suddenly descend into a more or less parietic section below it, or the intussusceptum may be drawn into the intussusciens by a more active peristaltic action of the latter. Among other exciting causes may be mentioned a tumor of the bowel, general lack of tone, such as may occur from a state of poor nutrition, chronic diarrhea, or constipation. According to some, the greater frequency of the condition in boy babies, is dependent upon the reflex irritation of an adherent foreskin.

#### PATHOLOGY

The postmortem appearance of intussusception will show an elongated tumor, usually on the left side of the abdomen. The invagination will produce the appearance as though the intestine was abnormally short. Above the point of obstruction the intestine is usually dilated and filled with gas and feces, while below it is generally empty, or in some instances may contain a small quantity of bloody mucus. The sheath is distended and frequently ulcerated, and there may be symptoms of general or local peritonitis. The intussusceptum is described as being commonly of a deep-red color, unless gangrene has set in, when it is black or greenish black in hue. The surrounding

serous layers become in a short time so united under inflammatory action as to prevent reduction of the invagination. Perforation of the intestine from ulceration may take place, or a stricture occur as a secondary consequence of inflammatory action.

**Symptoms.**—The most prominent symptom of intussusception is pain. The pain is generally of sudden onset, beginning in the region of the umbilicus, and is at first paroxysmal and very severe—really agonizing. Occasionally it may radiate from the back forward. As the disease progresses the paroxysms of pain become merged, until finally it is constant. Each onset of pain may be accompanied by a discharge from the rectum of a quantity of mucus and blood. Prior to such discharges, however, the lower bowel may have been emptied, and so one or two *normal stools* may have passed. In infants and young children the symptoms are more acute than in older children, and the pain is more paroxysmal in type. Between the attacks the child is quiet, but while the paroxysm is in progress the patient screams and assumes the characteristic position of intense abdominal pain—namely, the dorsal position with the thighs drawn up on the abdomen and the legs flexed against the thighs. Nausea and vomiting appear early as symptoms, and are frequently most exhausting. The vomited matter consists first of the contents of the stomach, but later frequently becomes stercoraceous. Rectal tenesmus of severe type is generally present. The abdomen is distended and tympanitic, and on the left side, usually in the iliac region, will be found a tumor which is often quite well marked and is associated with a corresponding flattening on the right side of the abdomen. The tumor is well described as “sausage-shaped.” Extreme tenderness is manifested over the seat of the tumor, although the entire abdomen is tender to the touch. This is especially the case in the later stages of the disease or when peritonitis has set in. The pulse is quickened and, as the condition progresses, has all the characters of the pulse of abdominal inflammation. The temperature is seldom elevated at first, and is more frequently subnormal, but after inflammation (local or general peritonitis) has occurred, it is raised to about 101° to 103° F. (38.3° to 39.4° C.). The mind is clear; the countenance, while generally tranquil between the attacks of pain, will, if the condition continues for some time, assume the peculiar pinched expression always seen in peritoneal inflammation. The amount of tympanites is only moderate, and indeed may not be present. There is only one sign which is frequently noticed—namely, the depression in the right iliac fossa. This is sometimes known as the sign of Dance (*signe de*

Dance). The symptoms may subside gradually if the attack progresses to a favorable termination. A rectal examination should always be made, preferably under anesthesia, for the tumor may be readily felt or the intussusceptum itself palpated. One of us had such an examination result most fortuitously, *for he inadvertently relieved the condition*. When the pain ceases suddenly, it is usually a sign that gangrene has occurred in the constricted portion of the bowel. This has been followed by a discharge of the gangrenous portions and a subsequent recovery. Very frequently a sudden break in the temperature, accompanied by cessation of pain and rapid, thready pulse, points to the onset of collapse and death. Partial or entire suppression of urine may occur.

**Diagnosis.**—Intussusception can be differentiated from: (1) Colic; (2) enteritis or dysentery; (3) fecal impaction; (4) appendicitis. From *colic* it may be distinguished by the vomiting, particularly when this assumes the stercoraceous character. The special diagnostic point, however, is the tumor on the left side of the abdomen. The bloody mucous discharge is present in intussusception and not in colic. The general severity of the symptoms seen in intussusception will also aid in the diagnosis. From *enteritis* intussusception is to be distinguished by the presence of bloody discharge in the latter, the absence of fever, and the presence of a tumor. In dysentery there may be a blood-streaked diarrhea, but the violent attacks of pain and the general symptoms of peritoneal involvement are absent. *Impaction of feces* can be diagnosticated by the total absence of the general symptoms of intussusception, and by the fact that the tumor produced by the greatly distended bowel is on the right side of the abdomen, and by rectal examination. Scybalous masses should also be detected along the course of the sigmoid flexure. *Appendicitis* may be distinguished by the character and location of the pain, the presence of tenderness at McBurney's point, right rectus spasm, constipation, and leucocytosis. The general abdominal symptoms of an appendicitis which has existed for some time are generally those of suppurative peritonitis.

**Prognosis.**—The outlook for a fortunate termination without operation is very unfavorable. In this class of cases the best chance for recovery is when the invaginated portion of the intestine sloughs and is passed by the rectum. According to Treves, the mortality in 133 cases in which original operations were performed for invagination was 72 per cent. In cases where reduction was tried and was found to be easy, the mortality was 30 per cent.; or in difficult cases of reduction it was placed at 91 per cent. The best prognosis is in those

cases which are operated upon early in the disease. In chronic intussusception the prognosis is very gloomy.

**Treatment of Intussusception.**—When unrelieved, the condition is so rapidly fatal in children that no delay whatever should be permitted after the diagnosis is established, and prompt attempts made to reduce the invaginated bowel. Even a few hours' delay is unwise. It is best to view intussusception as a surgical malady. An anesthetic should be given, the hips raised up on a pillow, and an injection of warm water given with a fountain syringe. The greatest gentleness must be exercised for fear of rupturing the gut, and the force of the column of water regulated by raising or lowering the bag of the syringe, each two and a half feet of elevation representing about one pound of pressure to the square inch. Injections of warm water containing a teaspoonful of salt to the pint, or of warm olive oil are also beneficial in aiding the reduction of the intussusception.

Inflation of the bowel with atmospheric air administered through a long rectal tube and bellows may be used with advantage in some cases. The inflation of the bowel with hydrogen gas or carbonic acid gas has been recommended, the former by Senn, the latter by Ziernsen, Libur, and Jate. Caution must again be exercised.

If this is not successful, the child may be inverted and gentle manipulation of the abdomen attempted. This measure should never be used after the first twelve to twenty-four hours, as by that time, especially if the constriction of the bowel be very acute, softening of the coats will have occurred and the danger of rupturing it is very great.

If this is not successful, immediate abdominal section must be performed and the invagination reduced by direct manipulation.

All of these methods of treatment have their greatest amount of usefulness in the first twelve or twenty-four hours of the intussusception. The patient should be allowed no food until reduction is accomplished. No laxative or cathartic medicine should be given by the mouth. Alcoholic stimulants are indicated when the patient is in danger of collapse.

After that time the efforts of the medical attendant should be directed to sustaining the patient, with the hope that the process of sloughing and evacuation of the strangulated portion will occur. During this time the administration of opium is of great importance, and the patient should be kept in the Fowler position. The patient's thirst may be relieved by small quantities of cracked ice, or, better still, by rectal injections of water. To relieve the vomiting, the stomach may be washed out by means of the stomach-tube.

**Operation for Intussusception.**—The abdomen is to be opened in the median line and attempts made to reduce the intussusception, provided the condition of the bowel warrants the belief that the integrity of the intestinal wall is not destroyed. If the operation be done early—within twelve to twenty-four hours—and the constriction be not extreme, this may be accomplished. If, however, the bowel shows evidence that its circulation has been materially interfered with and that there be any suspicion that its vitality is lost, it should be brought up and attached to the wound in the abdomen, an artificial anus thus being established. It would seem inadvisable to attempt an immediate resection of the damaged portion of the bowel, as at this time children are always in profound depression and their vitality is low. It is, therefore, best to relieve the immediate symptom in this way, leaving it to a subsequent time to complete the closure of the artificial anus.

### Volvulus

The symptoms produced by volvulus, or twisting of the bowel, will depend to a certain extent on the part of the bowel in which the twisting has occurred. Thus, in cases where the constriction is in the small intestine, vomiting will occur early and be persistent and severe. Wahl's sign, which he considers diagnostic, consists in a circumscribed area of tympanites corresponding to the location of the twisting. It is caused by the distention of the twisted loop of intestine by gas. Volvulus is most likely to occur in the lower portion of the ileum and the sigmoid flexure of the colon, and as compared with other forms of intestinal obstruction it is rare. Its causes in many cases are obscure. It has been ascribed to an accumulation of intestinal contents above the constricted portion of the bowel, or in some instances may be produced by adhesions of a loop of intestine to a portion of the omentum. Barring fetal volvulus (see malformations of the intestines), this condition is so very rare in children that it is practically unknown.

### HERNIA

Inguinal hernia in children may be divided into three classes. (1) The congenital; (2) the funicular; (3) the encysted, or infantile:

1. In the congenital form a loop of intestine makes its way through the open funicular process. In cases where it passes into the scrotum it will frequently envelop the testicle.

2. In the funicular variety the hernia passes down the open canal, but does not envelop the testicle because of the closure of the funicular process above the testicle by the tunica vaginalis.

3. The infantile form is the rarest of the three varieties. In this the funicular process is closed above, but not below, and the intestine is encased in a pouch of peritoneum, forcing its way into the process and thence descends (Rotch).

It is probable that the majority of inguinal herniæ, even those occurring in adult life, are of the congenital variety.

**Symptoms.**—In male children a soft round tumor will be felt extending from the internal abdominal ring into the scrotum. By proper manipulation, with the child in the supine position, this tumor can be easily made to disappear as the gut passes into the abdomen through the abdominal ring. In doing so it will give the characteristic gurgling sound. The testicle may be difficult to outline, but it will be found above and behind the tumor. In female children the labium majus will be distended by the loop of intestine, which is generally quite easily reduced. Usually in both sexes the tumor consists of intestine alone, but occasionally some mesentery may descend with it.

**Diagnosis.**—The condition with which hernia is most likely to be confounded is hydrocele.

#### HYDROCELE

1. Translucent by transmitted light.
2. Always dull on percussion.
3. When reduction is possible, the fluid passes back slowly and noiselessly.
4. No impulse on coughing.
5. The ring is empty.

#### HERNIA

1. Is opaque.
2. Always resonant.
3. The hernia passes back quickly and gives the characteristic gurgling sound.
4. An impulse can be felt when patient coughs.
5. The ring is filled with the neck of the tumor.

It should be remembered that these two conditions are occasionally associated.

**Prognosis.**—The outlook for cure in hernia is very good providing the child wears a properly fitting truss until the ring has had a chance to close. Strangulation is much rarer in children than in adults; but we have seen it in early infancy.

The **treatment** should consist in the prevention of constipation by the use of proper foods and medicines. The hernia should be reduced and a proper truss fitted.

**The Radical Cure of Inguinal Hernia.**—For many years past various operations have been devised for the radical cure of inguinal hernia, but failures were frequent and the risk to life was too great

for their general acceptance. We now have, in the methods of Bassini and of Halsted, which are similar in principle, the means for effecting a radical cure of this distressing and dangerous affection.

These methods consist in freely opening the inguinal canal, ligating the sac, transplanting the spermatic cord, and bringing it out at a point above the internal ring; then, by means of buried sutures, firmly uniting the tissues and thus obliterating the normal inguinal canal.

The results following these operations in many thousands of cases in the hands of hundreds of operators throughout the world have been so uniformly successful, both as regards final and permanent results, in effecting radical cures, that we are no longer justified in refusing our little patients the benefit of this method of treatment.

While it is true that a large majority of the cases of inguinal hernia in children recover simply by the aid of a properly fitting truss in the course of one or two years, persistence in the use of this method should not be carried beyond this period of time.

It is not fair to permit a child to suffer all the discomforts of wearing a truss for many years, and during this time risk the grave dangers of strangulation, when it lies in our power, to effect a certain and permanent cure.

The mortality, when modern methods of antiseptic cleanliness are carried out, is almost nothing—far less, indeed, than risks of strangulation which the child runs with even a perfect truss.

### Femoral Hernia

In this form of hernia the intestine passes under Poupart's ligament and makes its way through the femoral canal, showing itself directly under the saphenous opening. In femoral hernia the tumor is always on the outer side of the spine of the pubic bone. This form of hernia is much rarer in children than is the inguinal form, and is always acquired, never congenital. It is much more common in female children than in males. The treatment consists in the wearing of a suitable truss—and this failing, as it often does, of a radical cure by operation.

**Umbilical hernia**, the most common form in early life, has been dealt with in another section.

## DISEASES OF THE RECTUM

### PROLAPSE OF THE RECTUM

**Etiology.**—Prolapse of the rectum usually arises from a condition of atony following protracted diarrhea. It is not uncommon

also in attacks of diarrhea following prolonged periods of constipation. Its next most frequent cause is the straining at stool so often encouraged in constipated children. Violent attacks of coughing, as in pertussis, may produce prolapse of the rectum. The condition is not uncommon in children affected with chronic intestinal catarrh, and is sometimes seen following the violent diarrhea of milk infection.

**Symptoms.**—The bowel can be recognized, appearing as a tumor through the anus; the mucous membrane is usually of a bluish-red color, from the interference with its circulation.

The **treatment** consists in the removal of the causes. Constipation should be prevented or relieved by the use of enemata or other methods before mentioned; later the bowel movements should be kept liquid or semiliquid by the use of laxatives. The prolapse should be replaced and a return prevented by the use of rectal injections or suppositories containing some astringent, such as vinegar, alum, tannin, etc., and the child should evacuate the bowels while lying down, using pads or cloths to collect the feces.

The tone of the intestine should be restored as much as possible by the use of cold-water injections and by the continued use of astringents and tonics, such as strychnin or nux vomica. Occasionally it is necessary to keep the bowel in place by the use of a pad and **T**-bandage.

The best and most satisfactory method for treating surgically prolapse of the rectum is by linear cauterization of the mucous membrane of the prolapsus, extending well up the bowel, but great care must be exercised not to interfere with the sphincter muscle and not to cauterize too deeply. The actual cautery should be used. This should be tried in all cases before the more radical operation of excision be undertaken. This latter procedure should only be performed by a surgeon of wide experience, and if the peritoneum is opened, the danger to life is materially increased.

#### RECTAL POLYPI

Polypoid tumors of the rectum may appear either in the pedunculated form or as simple hypertrophic growths of the rectal mucous membrane. They are not at all uncommon during the early years of life; in fact some authorities think they are more usual then than at any other period. The characteristic symptom is hemorrhage, which may occur at any time, but is increased during evacuation of the bowels, at which time there is also great pain. Rectal polypi are of various

sizes and, according to Rotch, may be of the myxofibromatous or of the adenomatous variety.

Their treatment consists in removal either by section or by twisting them off.

### FISSURE OF THE ANUS

**Etiology.**—This condition may be produced by bungling attempts at introducing the nozzle of a syringe into the rectum, or by irritation caused by the presence of intestinal parasites. The most frequent cause is a slight excoriation of the parts resulting during the expulsion of a hardened mass of feces.

**Symptoms.**—When the fissure is of recent origin, the appearance is that of a small slit at the mucocutaneous orifice of the anus. When the condition has lasted for some time, the fissure may appear as an ulcer with indurated margins. The fissure may contain pus and even blood, both of which will be discharged during the process of evacuating the bowels. The amount of pain is very considerable: often so severe as to prevent the child from making attempts to move the bowels, and from this arises constipation, which aggravates the condition.

The **prognosis** is usually good, under appropriate treatment.

**Treatment.**—The constipation should be overcome by the use of suitable remedies, and particularly by laxative enemata. The local treatment consists in touching the fissure with a solution of nitrate of silver after having washed it out with some nonirritating antiseptic solution. In very severe cases it may be necessary to stretch the sphincter, and this should always be done when the condition approaches chronicity.

### ISCHIORECTAL ABSCESS

**Etiology.**—This condition arises most commonly from inflammation of the rectum. Such abscesses are often tuberculous, and one should carefully search for tuberculous disease in other organs. Traumatism may also be a cause. In the majority of cases the abscess is small, circumscribed, and superficial. The prognosis is generally good.

**Treatment.**—The treatment consists in the evacuation of the abscess under antiseptic precautions. If it is tuberculous, more radical measures are necessary. The incision must pass into the

bowel and through the sphincter, and all pus-pockets must be carefully explored and curetted.

### HEMORRHOIDS

Hemorrhoids during childhood have the same characteristics as in later life. The most common cause is chronic constipation. They are rare in children under three years of age, and much rarer throughout childhood than in adult life. The treatment is the same as in hemorrhoids occurring in adults.

### INCONTINENCE OF FECES

Inability to control the bowels is not infrequently seen in children whose powers of resistance are lowered from various causes. A nervous incontinence, similar to enuresis, has been described in neurotic children. Incontinence of feces also appears as one of the symptoms in the paraplegia of certain nervous diseases or any injury to the spinal cord.

The treatment should be directed to the cause. When the trouble is local, cure may sometimes be effected by the use of ergot given by the mouth or by rectal suppositories. Strychnin is a valuable corrective to the lack of tone.

### PROCTITIS

Three forms of proctitis, or inflammation of the rectum, are generally described—namely: the catarrhal, membranous, and ulcerative. In the catarrhal form the pathologic changes are those usually found in the same condition in other parts of the intestinal tract. The mucous membrane is swollen, of a deep-red hue, with increased secretion of mucus. In many cases the mucous membrane bleeds easily. When the condition is of long standing, white or yellowish-white ulcers are to be found along any portion of the rectum, but their most frequent site is immediately inside the sphincter.

**MEMBRANOUS PROCTITIS; DIPHTHERIC PROCTITIS.**—It is probable that the great majority of these cases are due to infection by streptococci, although a certain number occur during a general infection of diphtheria. The pathologic changes are the same as those found in the condition known as membranous enteritis.

**ULCERATIVE PROCTITIS.**—This form of inflammation of the rectum is characterized by the presence of ulcers occurring along the mucous

membrane. These ulcers may be superficial or deep, and are usually multiple. The condition is commonly caused by the progression of a catarrhal inflammation into the ulcerative type. The depth of the ulcers may vary rather considerably. They may be shallow, involving only the mucous membranes, or may extend deep down into the muscular coat. In the latter cases they very frequently become chronic and are of indefinite duration. Ulcers of the rectum may also be of tubercular or of syphilitic origin.

**Etiology.**—Proctitis may be produced by an extension downward of an inflammation affecting the upper part of the large intestine; or by traumatism, as from careless efforts at the introduction of a nozzle of a syringe. Intestinal parasites may produce it, and in female children it may be caused by an extension of a gonorrheal inflammation from the vagina or urethra or by direct infection through the anus. It occasionally follows or appears during the course of the acute infectious fevers. A very frequent cause is the use of irritating drugs, used either by injections or suppositories given with the intention of relieving constipation. Holt states that he has seen it produced in an infant a year old by the prolonged use of glycerin suppositories.

**Symptoms.**—In the catarrhal form the stools are increased in frequency and in the force by which the contents of the bowel are discharged. In this variety there is also usually a discharge of mucus, either in a liquid condition or in the form of a cast, sometimes mixed with traces of blood and preceding the discharge of the fecal contents of the bowel. True hemorrhage is rare. Tenesmus is nearly always present. Owing to the irritating character of the mucus, the external parts surrounding the anus become inflamed. Prolapse of the mucous membrane is not uncommon. The symptoms of the membranous form are very much like those of the preceding variety, with the exception that the discharges from the bowel contain a larger quantity of pseudomembrane.

The most marked symptoms of ulcer of the rectum are pain and hemorrhage. The pain is usually severe, and is referred to the region of the anus or coccyx or the surrounding parts. The hemorrhage is seldom severe, although occasionally quite a large quantity of blood may be passed. It is usually clotted and will accompany every movement of the bowels. In chronic cases more or less pus may be found with each stool.

**Treatment.**—The indications for treatment are to keep the patient at rest, to aid the digestion as much as possible, and to relieve the local condition. When the pain is severe, suppositories or injections of

starch water and opium should be used, or cocain may be brushed over the surface of the mucous membrane. Cocain may also be applied in the form of a suppository. Bland, slightly alkaline injections are of great use. They should consist of starch or lime-water or 1 per cent. saline solution. When the rectal discharges are highly acid, the enemata may be made alkaline, or alkalies may be given by the mouth. In order to decrease the quantity of the evacuation injections of saline fluid should be given, and these followed by enemata containing tannic acid in a strength of ten grains to the ounce. Such strong solutions are very irritating to some rectums, however. Nitrate of silver solutions (1-2000) are often very useful. A 1 per cent. solution of hamamelis has also been recommended for this purpose. The same treatment as is used for the catarrhal form will apply to the membranous variety. Ulcers of the rectum are in many cases obstinate and slow in yielding to remedies. The indications for treatment are rest in bed, a bland diet largely made up of milk, and the injection, two or three times a day, of boric acid solution. Local applications of a solution of nitrate of silver of the strength of one or two grains to the ounce may be applied to the ulcer after the bowels have been well washed out. We believe that such cases should always be treated by a rectal specialist. Opium should be given if the pain is excessive.

### INTESTINAL PARASITES

The parasites which most frequently infest the intestinal tract of infants and children are the oxyuris vermicularis, or the pin- or thread-worm, and the ascaris lumbricoides, or round-worm. Two species of tapeworm are also recognized, the *tænia mediocanellata*, or beef tapeworm, and the *tænia solium*, or pork tapeworm. The *trichocephalus dispar* is also occasionally met with. None of these parasites is peculiar to infancy or childhood. The pin- and round-worms are, however, so frequently found during the earlier years of life that they are classed among the parasites peculiar to children. The tapeworm, also, is not infrequently found in intestines of children, but, as a rule, it does not appear in as young children as do the other intestinal parasites. In addition to these common parasites of children in all climes, the "hook-worm" has assumed such great importance in the Southern States that it must receive consideration.

OXYURES VERMICULARES are small worms of a pinkish-white color and fusiform shape, which inhabit the rectum, the large intestine throughout its entire length, and the lower part of the small intestine. The female of this parasite, which exists in greater numbers than the male, measures about half an inch in length, the male being about

one-half as large as the female. The female is to be distinguished by its more slender and tapering shape, as well as by its greater length. To the unassisted eye they somewhat resemble small pieces of white thread; hence their name. With a low-power objective the uterine ducts in the female will be seen to contain numerous ova, these being ovoid in shape, about  $0.053 \mu$  in length and  $0.028 \mu$  in breadth. The most common symptom denoting their presence is a constant itching around the anus. This irritation increases at night, and particularly when the child is in bed. In girls the same irritation appears at the entrance of the vagina, and the parasites themselves may be found here as well as on the buttocks near the anal opening. From the irritation caused by their presence a true vaginitis of catarrhal type may be set up.

**The Mode of Infection.**—The original ovum is probably carried to the mouth by toys, food, vegetables, drinking-water, or on the fingers of those previously infected, and each ovum brings forth a fully developed worm. Very soon the irritation produced by the worm causes the child to scratch about the anus, and numerous ova are thereby lodged under the finger-nails, to be taken with the food. It is a curious fact that the presence of the worm does not cause irritation unless it is in the lower part of the rectum near the anus. Other than the intense itching there are absolutely no characteristic symptoms which point to the presence of pin-worm. The parasites themselves may be frequently found around the anus and about the vaginal opening of little girls. When the vagina becomes infested with them there is set up a vaginitis, accompanied by a very free discharge of mucus, which may occasionally be tinged with blood, caused by the continued scratching. From the same cause the anal region and buttocks may become excoriated, and later a genuine eczema may make its appearance. If the cause is long continued, the appetite becomes capricious, the child loses flesh, becomes nervous, has dark circles under the eyes, does not sleep well at nights, and, as in all other species of digestive irritation, is very apt to pick at the nose. Various other nervous symptoms may possibly appear, such as chorea and attacks of fainting. The diagnosis should be based on the discovery of the worms themselves. There are very few diseases with which they could be confounded, except chronic gastro-intestinal catarrh, which has more clearly defined symptoms. An enema given will scarcely fail to bring away enough of the parasites to make the diagnosis plain. In female children the irritation of the vagina may be confounded with specific vulvovaginitis, but the character of the discharge and the finding of the parasites will not long leave the diagnosis a doubtful question.

**Treatment.**—The first indication for treatment is to cleanse the bowels thoroughly of the worms and the mucus that surrounds them. This is best done by the administration of from one to three grains of calomel, in combination with from one to three grains of resin of scammony (dose for a child of eight years). Remedies such as spigelia, santonin, and others of the same class are often indicated. These should be given by the mouth in combination with calomel or any agent, such as sulphate of magnesia, which will produce free watery movements. Once a day the rectum should be washed out with a copious enema of cool, weak soapsuds, and a soft-rubber catheter should always be used in giving the enema instead of the ordinary syringe nozzle, in order that the liquid may flow as high up in the bowel as possible. A very useful formula to be given by the mouth is the following:

R	Calomel.....	1/2 to 1 grain
	Bicarbonate of soda.....	1 grain
	Santonin.....	1/2 to 1 grain
	Podophyllin.....	1/20 grain

SIG.—To be repeated every night for two or three nights (for a child nine to twelve years of age).

When relaxation of the rectum is present, injections of common salt solution, quassia, alum, tannin, etc., may have a certain effect in relieving this condition. Dr. Charles W. Townsend of Boston recommends the injection of one dram of sulphate of iron to one pint of infusion of quassia for this purpose. In order to prevent the transferring of the ova by means of the finger-nails, the hands of the patient should be carefully washed in soapsuds after each movement of the bowels, and the parts around the anus should be well cleansed with soap and water and smeared with an antiseptic ointment. When eczema is present, the following ointment may be used, both for its antiseptic properties and to allay the irritation caused by the parasites:

R	Boric acid.....	1 dram
	Acetanilid.....	20 grains
	Oil of rose.....	5 drops
	Vaselin.....	2 ounces.

It should be remembered that all means for the transference of the ova should be removed as far as possible. The bedclothing should be boiled and washed—in fact, all the clothing and furniture used by the patient should be carefully antisepticized.

**ASCARIS LUMBRICOIDES** (*Round-worms*).—The females of the round-worms measure from four to twelve inches in length, and are of a gray-

ish or reddish-white color. They somewhat resemble earth-worms, from which, however, they can be distinguished by their color and the fact that the earth-worm has plainly marked segments which can be seen on close examination. The ascaris is occasionally found in the small intestines in large numbers. They are much given to wandering about, and may pass into the stomach, into the large intestine, or gall-bladder. It is said that in rare instances they have been found in the appendix, the esophagus, in the pharynx, mouth, nose, or even in the lungs. They have passed through typhoidal ulcers into the peritoneal cavity. The female can be distinguished from the male by its greater size; it is also more slender than the male, the tail being straight and tapering, while that of the male is curved and blunter. The ova are produced in immensely large quantities and are passed off in the feces, in which they can without much difficulty be discovered by examination with a low-power objective. They are oval in shape, about  $1/400$  of an inch in length, and have thick, transparent coats, within which the dark granular contents of the ovum can be seen. The ova, which are extremely resistant to destruction by external influences, are usually taken in by the mouth from toys, food, drinking-water, or the dust which adheres to the fingers. Children, in crawling or playing around a room, and thus bearing on their hands a quantity of dust which in many instances has come from the street, may thus carry the eggs of the parasite into the system. It is certain that ascarides are less frequently found among the better class of people in whom the laws of cleanliness are more strictly carried out than among the poor and those of uncleanly habits. They are much rarer now in all classes than they were a score of years ago. Constant bathing, with proper care of the finger-nails of children, and seeing that their food is well cooked, will do much to prevent infection by these parasites. The round-worm is much more frequently found in children after the third year than in infants or adults.

**Symptoms.**—It is certain that in many cases large quantities of these worms may exist for a long time within the intestinal tract without producing any symptoms at all. As the majority of intestinal parasites are found in children whose digestions are habitually weakened, the lining membrane of whose intestines are continually coated with mucus, in which these parasites thrive, the majority of the symptoms usually ascribed to the presence of worms are due in reality to a chronic state of gastric or intestinal catarrh. The classic symptoms which parents will usually expect us to associate with the presence of worms are: A capricious appetite, or in some cases a ravenous desire for

food, unattended with any increase in bodily weight, or in some cases an absolute decline of it; irregular feverish attacks; disturbed sleep and bad dreams, accompanied by grinding of the teeth; pain in the stomach after taking food, and picking at the nose, and very frequently the passage from the bowels of large quantities of mucus, or, where the parasites are actually present, a number of these or their ova. It will be seen that none of these symptoms described is in any way characteristic, except the passage of ova or parasites themselves any or all of the other symptoms may be found in most forms of intestinal catarrh. It cannot be doubted, however, that a number of reflex nervous symptoms are, in susceptible subjects, caused by the presence of intestinal parasites, and probably every physician has met at least one or more such cases, in which symptoms of severe nervous disturbance have been relieved by completely clearing these parasites from the bowels. A very interesting case has been reported by Townsend from the records of the Boston Children's Hospital, where a girl four years old, previously in good health, was attacked by convulsions and nervous tremors, which were distinctly traced to a large number of round-worms in the intestines. Her condition immediately improved as soon as the intestines were thoroughly cleared of these worms, and the symptoms returned as soon as a fresh quantity of the parasites were generated. There is also some danger of occlusion of the intestines, due to a simple mechanical obstruction caused by large masses of worms; thus, in Hillyer's case, in which a weakly child of five and a half years was attacked by severe abdominal pain, and upon being given a dose of oil vomited a round-worm. The child died on the following day, and at the autopsy the ileum was found occluded at a point fifteen inches above the ileocecal valve by "a tightly wound ball composed of eight round-worms; forty-two worms in all were found in the intestines. Below the obstruction the intestine was empty, while it was distended above." As previously remarked, round-worms are such travelers that there is no telling where they may not go and set up an irritation by their presence; thus they may penetrate the cystic or common bile-duct and cause jaundice by stopping the flow of bile. Abscess of the liver has been known to occur from their having penetrated this organ. They have also been known to penetrate the trachea, or even the lung, causing strangulation or pulmonary gangrene.

**Diagnosis.**—As the symptoms are so very unreliable, a positive diagnosis can only be made by microscopic examination of the feces. With a low-power objective, one which magnifies, say, about 320 to 350 diameters, the eggs will be easily recognized. Those of the round-

worm are to be distinguished from the oxyuris, the former being larger and rounder, while those of the oxyuris or pin-worm are sharp, smaller, and more oval. From tapeworm the ova of the ascaris are to be distinguished by the fact that the latter are rounder; we have, too, when tenia are present, the expulsion of the segments.

**Treatment.**—The most successful treatment consists in the administration of santonin, usually combined with calomel or castor, oil. Of all the drugs given to cause the death and expulsion of worms, santonin is probably the best. It must be remembered, however, that it is an extremely poisonous drug and must be administered with great caution. The toxic symptoms are gastro-intestinal irritation, muscular tremor, and a sensation as if the patient were looking through yellow glass. If the poisoning continues, there will be dizziness, extreme dilatation of the pupils, convulsions, and finally loss of consciousness. Santonin is best combined with calomel, from one to three grains of the former being given with one-half to one grain of the latter. Townsend, in the "American Text-book of Diseases of Children," gives the exact dosage of santonin as follows: For two years of age,  $\frac{1}{4}$  to  $\frac{1}{2}$  of a grain; at six years of age, 1 grain; and at twelve to fifteen years of age, 2 grains. In our own experience we have found these doses safe and reliable. The child should receive a dose in the morning and one at night, or in some cases three times daily. Some authorities recommend that a dose of santonin combined with calomel or oil should be given at night, to be followed in the morning by sulphate of magnesia or other saline. It should not be forgotten that as the patients who are most commonly infected with these worms are considerably below the average standing in bodily health, this condition should be attended to. Tonics should be administered, and the diet and general hygienic surroundings improved as much as possible. A diet containing a considerable amount of salted foods has been recommended by some authorities.

**TENIA (*Tapeworm*).**—The two species of tapeworm most commonly seen are in *tænia solium*, found in pork, and the beef tapeworm, or *tænia mediocanellata*. Another rare species known as the *bothriocephalus latus* is sometimes described. Two other rarer forms, known as the *tænia nana* found in dogs and cats and the *tænia cucumerina*, are so seldom seen in this country that they will not be more than mentioned. The length of the average tapeworm may be anywhere from twenty to fifty feet; they are of a white color, and receive their name from their resemblance to a piece of ribbon or tape. The *tænia mediocanellata* and the *tænia solium* are to be differentiated by the shape of the head,

The head of the *tænia solium* is rather pointed and contains four and also, to some extent, by the shape and size of the segments. sucking discs, surrounded by a circle of hooklets. The head of the *tænia mediocanellata* is much flatter, has four discs, but no hooklets. Both species are composed of a small head, about the size of that of a pin or a little larger, and an immense number of seg-

ments. Each segment is sexually complete in itself, or what is known as hermaphroditic.

**Diagnosis.**—The diagnosis can be made by finding the segments and carefully examining them through the microscope. The species can be determined by the difference in the shape of the head, already described. Microscopically, it will be seen that in the *tænia mediocanellata* the lateral branches of the uterus are finer and much more numerous than in the *tænia solium*.

### Mode of Infection and History of Development.

The eggs of the two principal varieties of tapeworm usually find their way into the intestines of their human hosts in the following manner: They are at first most probably contained in manure or fertilizer, and thence are taken into the stomach of the animals most commonly used



FIG. 26.—TÆNIA NANA, VON SIEBOLD. (After Leuckart.)  $\times 10$ .



FIG. 27.—HEAD OF TÆNIA NANA, VON SIEBOLD; WITH RETRACTED ROSTELLUM.—(After Leuckart.)  $\times 75$ .  
A. An isolated hook.  $\times 300$ .



FIG. 28.—EGG OF TÆNIA NANA, VON SIEBOLD.—(After Leuckart.)  $\times 300$ .

for food—namely, cattle and hogs. Having found their way into the stomach of the animal, the outer coverings of the egg are dissolved, and the embryo is set free. The embryo then pierces the stomach-walls, and, entering the blood current, is carried to any part of the animal's body, particularly the muscles, in which it buries itself, forming what is known as a cysticercus cyst. Usually not one but many of these cysts pass into the circulation of the animal at one time. Within the cyst the embryonic or larval tenia grows. These cysts are extremely tenacious of life, and they have frequently been known to

remain alive in the tissues of an animal for four or five years. Unless the tissue in which they lie dormant is taken into the human stomach, the embryo finally dies, and the cyst becomes calcified. If, however, the meat containing a living cyst is taken into the human digestive organs, the scolex grows rapidly into a mature tenia. The worm grows by the development of those segments nearest the head, these becoming mature as they progress further from the point of origin, and it is from these sexually mature segments that ova are given off.

The worm makes its home usually in the upper part of the jejunum, or at least it is here that the head is firmly attached to the mucous membrane by hooklets and suckers. The body, composed of a vast number of segments.

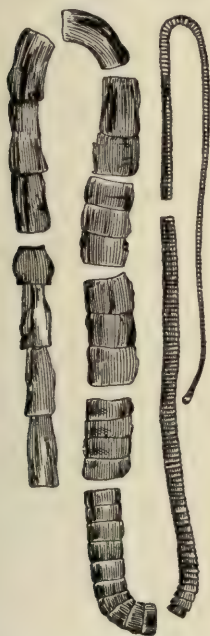


FIG. 29.—*TÆNIA SAGINATA*, GOEZE.—(After Leuckart.)

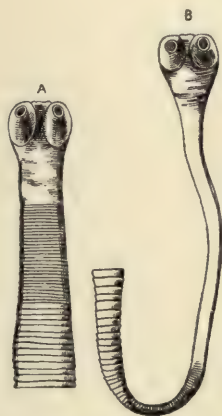


FIG. 30.—*CEPHALIC END OF TÆNIA SAGINATA*, GOEZE.—(After Leuckart.)  
A in retracted and B in extended state.

may extend for any distance along the intestine, depending on the length of the animal. Sometimes it may reach as far as the ileocecal valve. The *tænia solium* is usually found singly, while two or more of the *tænia mediocanellata* are often seen in a single intestine. It is quite possible—indeed, it occasionally happens—that the eggs of the tenia, after having been swallowed by man, pass through the coats of the stomach in the same way as in the animal, and develop a cysticercus cyst in any part of the body, especially in the subcutaneous or intermuscular connective tissue or in the eye and brain (Townsend). The cysticercus is extremely resistant to high temperatures, and it is usually by eating improperly cooked beef or

pork that the infection takes place. Babies may be infected through rare beef juice. Eating sausages made is a common mode of infection.

**Symptoms.**—The first and most common indication of the presence of a tapeworm is the finding of the segments or proglottides in the feces. Besides this, there is no regular sequence of symptoms denoting its presence any more than in other forms of intestinal parasites. Frequently there may be pain or sensations of discomfort, which are referred to the region of the umbilicus. The appetite may be great, but is accompanied by a gradual loss of bodily weight. When nervous symptoms are present, they are usually those previously referred to in the description of the other forms of intestinal parasites.



FIG. 31.—HEAD OF *TÆNIA SOLIUM*. ON THE RIGHT, EGG OF *TÆNIA SOLIUM*.—(Leuckart.)

**Treatment.**—The treatment should be aimed at dislodging, as quickly as possible, that part of the worm known as the head, and during the administration of remedies for this purpose the feces should be examined with the greatest care by the physician himself. The success of the treatment consists in the careful administration of any one of a very few drugs. Whichever one of these is used, the method of procedure must be thorough. A great deal of the success lies in the patient carefully carrying out the rules laid down for

him. The remedies most commonly used are pomegranate, or its alkaloid, pelletierin; pumpkin-seed, kousso, the root of the male-fern, turpentine, and cocoanut. Of all these, by far the most efficient is pelletierin. This may be given in the form of the tannate, which is an exceedingly efficient remedy, although expensive. The dose of this for an adult is from five to twenty grains; the dose for children, however, should be regulated according to age. The method of administration of this drug is as follows: A preparatory treatment, consisting of partial starvation, should be instituted for some hours before the teniacide is given. During this time small amounts of food which is principally digested in the stomach should be given. It has been recommended that early in the evening the child be given a bowl of beef-tea with half a slice of white bread. In a little while the patient should receive an enema and be put to bed. On the following morning a cup of beef-tea should be given, and an hour after breakfast a full dose of the anthelmintic administered, to be followed in an hour by a good active cathartic. Great care should be exercised that when the worm is

expelled it is not broken off and part of it left behind in the rectum. To prevent this it may be necessary to dilate the sphincter gently by means of a rectal speculum. Next in efficiency to pomegranate and its alkaloids is the oil of male-fern, *oleoresina aspidii*. The dose of this drug for a child of five years is a teaspoonful. It may be given in four doses of fifteen or twenty grains each a quarter of an hour apart.

## HOOK-WORM DISEASE

The hook-worm (*ancylostoma duodenale*, *Necator Americanus*, etc.) so named originally, from the shape of its copulatory bursa, is the cause of the now well-known disorder under discussion. It is responsible for much invalidism in Africa, Egypt, the Philippine Islands, Porto Rico, and our own far Southern States, etc. Allen J. Smith states that it costs this country more than tuberculosis does.

**History.**—The hook-worm was first discovered in the intestinal tract of the badger (Göeze, 1782), next in the intestine of the fox by Froelich (1789). It was the latter investigator who dubbed the parasites hook-worms (*haakenwurm*), and proposed the generic term *uncinaria* (Stitt). Later (1838), Dubini found these parasites in the intestinal tract of man, but it was not until the building of the St. Gothard's tunnel (1870) that its full importance in the production of anemia was understood.

**Etiology.**—The studies of Stiles, Allen J. Smith and Loos have made our knowledge of the *Necator Americanus* what it is to-day. The structural peculiarities of the American parasite, imported by negroes from central and southern Africa, are well portrayed in Fig. 31A.

**Mode of Infection.**—The ova in the human subject affected with the disease, are deposited upon the ground. Here they require heat and moisture for their further development, being killed by a temperature of 1° C. The rather sluggish larvæ develop upon the moist ground, or in pools of water, and even climb blades of grass. Bare-footed children are readily infected by them, the larvæ actually piercing the skin, and causing, in transit, the well-known "ground" or "water-itch." Having entered the subcutaneous tissues the immature organisms may be carried by the lymphatics to the lungs, or they may bore through vessel walls, and be borne directly by the blood stream. Having arrived in the lungs, and having been divested of their enclosing envelopes, they follow up the bronchial tree to the esophagus, where they are swallowed. Arrived in the intestine, the worms attach them-

selves to the intestinal mucosa. Here eggs are released, after a period of 50 days, and the curious life cycle begins anew. According to Stiles, direct infection, through the mouth, also takes place.

**Symptoms and Clinical Course.**—The prominent symptoms in affected children are anemia, stunting of physical growth, retardation of physical and mental development, and great lassitude. (It is an injustice to speak of them as lazy.) Ground itch, tibial ulcers, and in bad cases, dirt eating (pica) are other symptoms of importance.

The disease is seen in poor people principally, and in those living under bad hygienic conditions. The habit of going barefoot is fraught with menace in infected districts.

According to Stiles, the employment of such children in the mill may really spell their salvation, for here they cease to go barefoot.

**Diagnosis.**—To one acquainted with this rather clear-cut symptom-complex, the clinical diagnosis should not prove difficult; but the strong suspicion so aroused should be made a certainty by examination of the stools for ova. (The mature worms are not found.) Should the usual search prove unavailing, as it may in from 45 to 60 per cent. of cases, cultural methods should be employed. For the latter search, an island of superimposed filter-papers is built up in the centre of a large Petri dish. Water is then introduced until its level is flush with the surface of the island. Feces are smeared thickly upon the filter-paper. In six days the larvæ hatch out and swim around in the surrounding water. This method is successful in 99 per cent. of cases.

**Prognosis.**—This depends upon treatment; social conditions of the patient, his hygienic environs and subsequent prophylaxis.

**Treatment.**—Infected individuals should be taught not to defecate upon the ground, and sterilization of feces in wells should be employed. In infected districts, children should not go barefoot; nor should they play upon the ground. Under present conditions, in certain regions of the South (Georgia, etc.), the children of the poor are safer in the cotton mills than they are in the open (Stiles). For the infected, thymol is the best remedy; though every safeguard should be taken to prevent its absorption by the host. Alcohol and fats must be eliminated from the diet on the day that thymol is administered. Little food should be taken the night before. The drug should be administered in three successive doses, about one hour apart. The last dose should be followed promptly by a full dose of Epsom's salts. Should the first treatment prove unsuccessful, the same procedure may be repeated in a

week or two. The dose of thymol is differently given by different authorities; probably x-xv grains under five years, and xv-xx grains between five and ten years would represent safe and efficient dosage when divided into three parts as suggested. Other remedies employed are betanaphthol and oil of encalyptus; but thymol is the drug of choice.

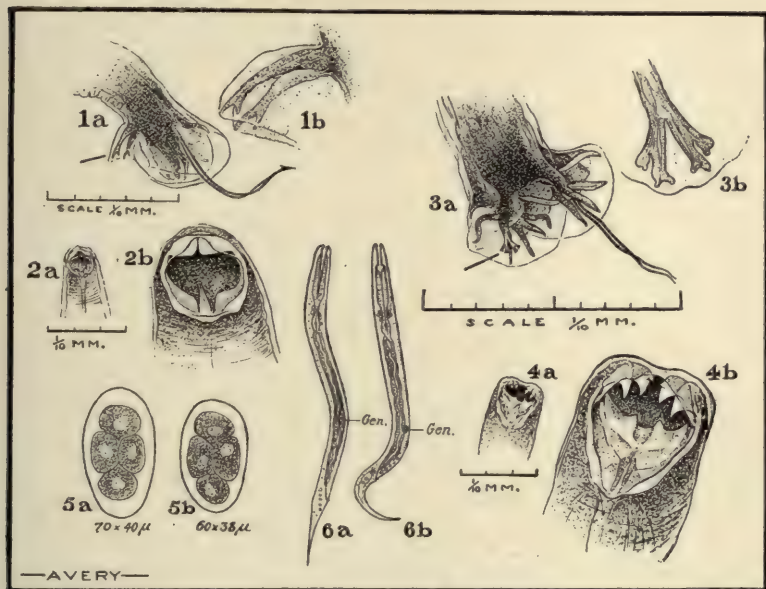


FIG. 31A.—1a, Copulatory bursa of *Necator americanus*, showing the deep cleft dividing the branches of the dorsal ray and the bipartite tips of the branches; also showing the fusion of the spicules to terminate in a single barb. Scale 1/10 mm. 1b, Branches of dorsal ray magnified. 2a, The buccal capsule of *N. americanus*. 2b, The same magnified. 3a, Copulatory bursa of *Ancylostoma duodenale*, showing shallow clefts between branches of the dorsal ray and the tridigitate terminations. Spicules hair-like. 3b, The dorsal ray magnified. 4a, The buccal capsule of *A. duodenale*, showing the much larger mouth opening and the prominent hook-like ventral teeth. 4b, The same magnified. 5a, Egg of *N. americanus*. 5b, Egg of *A. duodenale*. 6a, Rhabditiform larva of *Strongyloides* as seen in fresh fæces. 6b, Rhabditiform larva of hookworm in fæces eight to twelve hours after passage of stool. (Stitt.)

## CHAPTER VI

### DISEASES OF THE PERITONEUM

#### APPENDICITIS

It has become a custom, by the common consent of authorities, to apply the name appendicitis to inflammations in and about the vermiform appendix, notwithstanding the fact that inflammation of the cecum, typhlitis and perityphlitis, have received differential description in text-books. Clinical experience demonstrates that almost invariably inflammatory conditions in the right iliac fossa have a common origin in the appendix vermiformis.

Strictly speaking, appendicitis should not be included under diseases of the peritoneum, but under those of the intestines. As it is the most common cause of peritonitis, however, and as the student should always think of the latter as a possible dread complication or sequel of appendicitis, it has seemed best to us to describe the inflammation of the vermiform process in this place.

**Etiology.**—The causes of appendicitis may be conveniently divided into predisposing and exciting. Of the predisposing causes may specially be mentioned peculiarities of structure, both congenital and acquired. Appendicitis is more common in adolescence and early adult life (the periods of greatest dietetic indiscretions) than in later years, in those who are habitually or periodically constipated, and in those subject to intestinal catarrh.

Among the predisposing causes may be mentioned departures from the normal shape and situation of the appendix, together with conditions of feeble nutrition. These, combined with irregularities in the development of its mesentery, may tend to produce a condition of twisting, as has been pointed out by Broca. In 25 per cent. of cases, Vallee found the base of the appendix higher than in adult life, hence one might be misled concerning the McBurney point. On the other hand, the distal end (tip) is often located low down in the pelvis (Selter). The appendix is relatively longer in the child. "Only in less frequent torsions, or where the insertion is posterior, is its course toward the lumbar region. The amount of lymphoid tissue in the appendix has attracted much attention of recent years—the organ has actually been styled the 'appendiceal tonsil.'"

Accumulations of irritating or poisonous substances within the appendix, and especially collections of feces, may be considered as a predisposing rather than an exciting cause. Indigestion plays an important rôle as a causative factor. Certain authorities have drawn attention to the fact that appendicitis becomes more prevalent during epidemics of influenza. This seems to be more than a casual relationship, and we have seen at least three patients, in whom the disease immediately followed an attack of influenza. One of us has reported a case that occurred during measles. Appendicitis is also an occasional occurrence during the course of typhoid fever. Traumatism and the lodgment of foreign bodies such as seeds, pieces of hair, glass, pins, etc., are probably occasional causes. Exposure to cold and wet, traumas, the straining of the abdominal muscles, and abuse of astringent purgatives have all been described as exciting causes.

It has been found that the canal of the appendix may be obstructed by a calculus, to which the term "calculous appendicitis" has been applied. These calculi are usually formed singly, but occasionally three or four are seen together. They are made up of a stercoral base with a mixture of calcareous sulphates and phosphates; they also combine chlorids and sulphates. They are of a brownish color and variable consistency. Section of one of them generally shows stratification. They exhibit a slow, progressive development in the canal of the appendix. The lumen of the canal may also be obstructed by reason of a local affection causing swelling of its walls. As Rendu reports, the canal may also be obstructed by fibrinous formations. Lastly, two or more of these factors may be found associated in causing the obstruction.

**Bacteriology.**—Studies of the diseases of the vermiform appendix invariably demonstrate the presence of the *bacillus coli communis* in pure cultures or associated with other pyogenic or saprophytic organisms. Usually there is a mixed infection. From the degenerated condition of the cells of the mucosa of the appendix virulent types of malignant bacteria develop and may penetrate the peritoneal cavity, either through a perforation or, as shown by Klecki, through the lymph-spaces of the damaged intestinal walls.

**Morbid Anatomy.**—Inflammation of the vermiform appendix may be found in any of its classic stages; thus, there are recognized the catarrhal, ulcerative, and the gangrenous varieties. All of these are really similar processes, differences depending upon the source and virulence of the inflammation. Any one of these forms may be circumscribed or diffused. In the catarrhal variety the walls of the appendix

are found thickened and hyperemic. The submucosa and muscular coats are infiltrated by embryonic connective-tissue cells. The lumen of the tube is filled with the débris of inflammation, which is thus becoming narrowed, and finally, if the condition continues, the canal may become obliterated. The marked peculiarity of inflammation of the appendix is the rapid involvement of all its coats. The catarrhal stage may end in resolution or go on to obliteration of the canal and perforation of the tube. When ulceration occurs, the source is in the base of the muscular coat. The mucous and submucous tissues are for the most part destroyed. The ulcer may perforate the appendix or, if healing occurs, stricture of the tissues may ensue. Dilatation of the tube may occur beyond the point of obliteration. In the gangrenous variety, known by some as interstitial appendicitis, a rapid necrosis of all the coats of the gut takes place. In the majority of cases there is no foreign body contained in the lumen of the appendix; not infrequently, however, there is found a fecal concretion teeming with virulent bacteria. These, if perforation occurs, exude into the peritoneal cavity, starting up intense peritonitis. The organ may be partially or entirely necrosed—not infrequently the entire appendix is detached by sloughing. The general peritoneal cavity becomes walled off by fibrous adhesions, the result of extension of inflammation to the peritoneum. If perforation should occur before this happens, diffuse peritonitis results; otherwise the fibrinous exudate, by causing adhesions between the appendix, intestinal coils, and the abdominal walls, acts as a barrier to general infection. In most cases suppuration quickly follows the serous exudation, and a localized abscess is formed. This may break into the general peritoneal cavity or escape through the intestine, or form a fistula through the abdominal wall. Retroperitoneal abscesses occur when the perforation takes place along the line of attachment of adhesions. Hodenpyl calls attention to the fact that inflammation of the appendix may result from tuberculous or lymphoid ulcers, although this is rarely seen.

**Symptoms.**—Appendicitis may be divided into two varieties: the catarrhal, the phenomena of which are relatively slight, cases often escaping recognition, and perforative appendicitis, producing gravest appearances and effects.

Appendicitis in its incipient stage produces symptoms so obscure and varied in character that they are often either unrecognized or, if seen, their importance is not appreciated. The first symptom of the disease is pain. Its occurrence is generally sudden and unexpected and may be associated with a chill. The pain varies in intensity

from mild discomfort to extreme agony, and is usually intermittent. At the outset of the disease the seat of pain may be referred to any area of the abdomen (epigastrium, etc.) or, as sometimes happens, to the whole abdominal region. No matter where it begins, in a short time it is localized in the right iliac fossa or over the inflamed appendix. Tenderness quickly manifests itself on the right side, with its point of maximum intensity in the region of the appendix—that is, at a point near the outer edge of the right rectus abdominis muscle. Its position may be described as being near the center of a line drawn between the umbilicus and the anterior superior spine of the right ileum. This point is frequently called *McBurney's point*. It is incumbent upon us to recall that this point may be higher in the young child. Again, another point of tenderness may be felt which corresponds to the tip of the inflamed organ. In the event of the appendix occupying an anomalous position, this point of tenderness will be correspondingly changed. Resistance of the walls of the right iliac fossa becomes early noticeable. When a considerable amount of tenderness is present, the right rectus muscle becomes retracted and tense, so as to resist palpation. This sign, however, is not nearly so reliable as it is in adults. Often the whole region is readily compressible. The abdomen becomes distended and tympanitic, and a circumscribed swelling can be made out a little beneath the point of greatest tenderness. Gentle palpation will reveal a tumor of oval shape and some tenderness, the length being about two inches. We do not believe, however, that it is often possible to palpate the inflamed organ. One is more often able to feel it through the rectum than through the abdominal wall. What is often detected in abdominal palpation is the distended *caput coli*. Over the area of swelling the percussion-note is varied, this variation depending upon the proximity of the swollen appendix and its exudate to the abdominal wall. In the later stages of the disease fluctuation may, perhaps, be elicited in this area. To guard these tender points and give himself greater comfort, the patient lies in the dorsal position with the right leg drawn up. A rapid elevation of temperature is usual at the onset. In the beginning of the attack the fever may reach  $102^{\circ}$  to  $104^{\circ}$  F. ( $38.9^{\circ}$  to  $40^{\circ}$  C.), but later falls one or two degrees. Cases mild in type may exhibit temperatures of  $101^{\circ}$  or  $102^{\circ}$  F. ( $38.3^{\circ}$  to  $38.9^{\circ}$  C.) throughout their courses, and normal or subnormal temperatures are not seldom found in the severest cases. Continued high temperature points to suppuration, and a sudden fall, while indicating in a certain number of cases beginning resolution, not infrequently indicates perforation. Perforation is far

more prone to occur in children than in adults. It probably occurs in about 1/2 of the cases; often very early in the attack. The pulse is accelerated, the rapidity depending to a considerable extent on the height of the fever—a rapid pulse with a low temperature demonstrates that perforation has occurred. Vomiting is more or less constant from the beginning of the attack, and occasionally is a source of much distress. When the attack proceeds favorably, the vomiting usually subsides in the first day or two. When peritonitis occurs the vomiting returns and is persistent. The patient is usually obstinately constipated, although diarrhea is rarely present; and is sometimes seen in the late stages of a prolonged attack. The appetite is lost; the tongue is furred and covered by a brownish coat; thirst is generally present and may be intense. The urine is scanty and at times may be albuminous; in the majority of cases it is high colored. In the early stages of the disease the patient may feel a frequent desire to evacuate the bladder, probably because of the low position of the tip. When the attack proceeds to suppuration, the condition is indicated by rigors, sweats, and considerable exhaustion. In many cases, however, the formation of an abscess is indicated only by the continued elevation of temperature and an increased tenderness over the affected area. The tumorous mass in the right iliac region increases in size, but on account of abdominal distention can at times be demonstrated only with the greatest difficulty. Again a rectal examination combined with abdominal palpation may reveal the mass. General involvement of the peritoneum may occur at this time from rupture of the walls of the abscess precipitating its contents into the peritoneal cavity, or a general peritonitis takes place almost from the first, caused by the invasion of septic bacteria before local adhesions have been established. When this happens, the symptoms are those of a severe peritonitis, ending usually in collapse, death almost invariably following. A certain proportion of cases end favorably without treatment, but in these the symptoms are mild in type, with but little pain and a slight elevation of temperature, which gradually, or sometimes suddenly, falls to normal. When the disease has continued for some time and suppuration has taken place, the countenance assumes the characteristic pinched expression seen almost invariably in grave abdominal diseases. The fever may then assume a hectic type, and the patient passes into a genuine “typhoid state.” Infrequently in this class of cases the tumor in the right iliac region becomes boggy. The skin over it may be congested and slightly edematous. There may be pain in the right knee or ankle. Edema of the

right leg may occur. Leucocytosis furnishes valuable supporting evidence of the clinical diagnosis.

**Diagnosis.**—The recognition of appendicitis from its onset is of the utmost importance, and the symptoms most to be relied upon are the character and location of the pain, tenderness of the appendix itself, muscular tension of the right rectus muscle and the blood count.

*The diagnosis* should not be built upon the blood count: but it should occupy an important and appropriate position in the diagnostic structure. In most cases, leukocytosis (usually between 20,000–30,000) is present. If the other signs (physical examination, etc.) point indubitably toward appendicitis and leukocytosis is not present, the prognosis is grave. With an increased leukocytosis, even though other symptoms are ameliorating, pus formation should be suspected. In a severe case, where a sudden drop in the numbers of leukocytes is observed, the patient is probably overwhelmed with toxins.

Accepting the views of Richardson, sudden excruciating pain, becoming localized in the right iliac fossa, while of great diagnostic value in the recognition of appendicitis, indicates extension of inflammation to the peritoneum and perforation of the appendix rather than giving evidence of the incipency of the disease. Vomiting and constipation (rarely diarrhea) are early symptoms, but are of value only when associated with the more important diagnostic points enumerated. Palpating the appendix determines the amount of enlargement and also the degree of tenderness which may be present. Tumor and change in the percussion-note are not usually recognized before the third or fourth day. Appendicitis must be differentiated from: (1) Acute intestinal obstruction; this occurs with considerable frequency in children, especially infants, and not seldom becomes a possibility to be considered in making a diagnosis: When the obstruction is due to an intussusception, bloody discharges from the bowel are generally present, and the tumor, instead of occupying the right iliac fossa, is found either in the median line or more prominently in the left side. The possible detection also of the invaginated bowel by rectal examination will aid materially in the diagnosis. (2) When strangulation of the bowel is due to a twist or volvulus, the pain is not localized, as a rule; constipation is also more pronounced than in appendicitis. Moreover, in this form of obstruction, as well as in intussusception, the vomiting is apt to be stercoraceous and is persistent. Besides this, where a volvulus occurs the abdomen is generally distended. This condition is so rare in early life, though, that it need not demand much attention. (3) Obstruction of the bowel by col-

lections of feces in the cecum may cause a low grade of inflammation; this condition can be recognized by its gradual development, the boggy feel of the tumor, which can be felt as an elongated mass lying in a vertical direction. There is absence of a localized point of tenderness and pain, and almost never symptoms of perforation. The inflammatory symptoms are less severe. Similar masses (fecal) can also be felt in the sigmoid region, and are always present in the rectum. (4) Acute indigestion and enterocolitis are excluded by the absence in them of tumor, of localized tenderness, the different character of the vomited matter, and the continued diarrhea with mucous stools. (5) Hip-joint disease and tubercular peritonitis are mentioned as sources of possible error in making a diagnosis of appendicitis, but these can be excluded usually by strictly considering the history of the case, the symptoms, and general physical aspects of both the above diseases. However, it is to be remembered that either or both of these conditions may possibly coexist with appendicitis, in which case a diagnosis is of grave moment and requires much skill. (6) In girls at the age of puberty an abscess of the right ovary is quite difficult to differentiate from appendicitis; indeed, gynecologists have repeatedly called attention to the frequency of the association between appendicitis and disease of the right ovary. The diagnosis could only be settled by vaginal examination or rectal examination or, better, both. However, it is more common to find appendicitis in males than in females, and besides this the shape of the tumor, and the generally greater severity of symptoms of appendicitis, will aid in the diagnosis. It may often be impossible, without opening the abdomen, to arrive at a correct diagnosis. It is well to regard appendicitis as a surgical disease and to associate an able surgeon with the medical attendant at an early stage of the disease.

**CHRONIC APPENDICITIS.**—The terms recurrent and relapsing appendicitis have been used in describing the return of symptoms after the first attack has subsided. One attack predisposes to another in the majority of cases. The inflammatory process of the primary seizure may have entirely disappeared, but with the result of leaving the appendix extremely susceptible to the slightest irritation, so that now and then the patient will suffer from a recurrence of the disease. The symptoms of these relapses may be as severe as the original onset, but as a general rule they are milder in type; however, the possibilities are always grave. On the other hand, the primary inflammation may subside into a latent or subacute form, causing a constant discomfort to the patient. In these cases there is an exacerbation of symptoms at

short intervals. The term relapsing appendicitis has properly been applied to this latter form.

**Prognosis.**—Few conditions so well illustrate the weaknesses of the prognostic art. Most of the catarrhal cases will recover; but no one can tell when recurrent seizures may threaten. On the other hand, interval operations present an almost negligible mortality. With abscess formation the prognosis is bad unless an operation be done. The same may be said of gangrenous appendicitis. When general peritonitis has occurred the case is well nigh hopeless, but operation occasionally results in recovery.

**Treatment.**—As soon as appendicitis is suspected, the little patient should be put to bed and kept there in the Fowler position. Much less absorption of toxin takes place from the pelvic peritoneum than from the peritoneal surfaces higher in the abdominal cavity. Following Ochsner, food should be withheld for at least two days, and after this time, only peptonized milk should be given in very small quantities. The Murphy method of introducing fluid into the bowel drop by drop should be employed in all cases. Sometimes we employ it continuously, allowing 10 or 12 drops a minute to flow. At other times, we employ two hot (110°) colonic lavages daily, and employ the "drop method" for a period two or three times a day. Ringer's solution is employed in preference to normal saline. Hot wet compresses are usually applied locally. These are covered with oiled silk or muslin. Cold, however, frequently relieves pain more quickly. An ice-bag should not be employed continuously for days, and one should always watch for marked local congestion of the surface when ice is used locally. Our views concerning the employment of morphin and of purgatives have undergone many changes in the last two decades.

We still employ salines if we see the patient very early in the attack. Later, we employ morphin if suffering is very great. The morphin is given hypodermatically, usually in conjunction with atropin. In the presence of severe vomiting, calomel may be given in fractional doses, though again, morphin may be required to quiet it. Of greatest value, however, in the non-operative treatment of early appendicitis in children is starvation, the use of the Fowler position and the Murphy treatment.

The question of temperature is of comparatively little importance in determining the need for surgical interference, as it may continue high, even though the local pain and tenderness diminish.

A sudden relief from pain and a low temperature but high pulse-rate indicate perforation of the appendix and a probable infection of the

general peritoneal cavity. Sudden cessation of the pain is also often observed with the occurrence of gangrene.

Of course, a very large number of cases recover from the effects of the first acute attack by simple measures of treatment, but it is absolutely impossible to take any number of days or hours as a guide for the necessity for surgical interference. The amount of infection of the appendix may be so great that gangrene and perforation may occur as early as in from fifteen to twenty hours after the first signs of pain, while, on the other hand, the progress of the disease may be less rapid in its development, and several days may elapse before these changes exist.

When the symptoms persist, despite the medical measures detailed, the pain, tenderness, and resistance on palpation in the right iliac fossa increase rather than diminish, the pulse continues rapid or grows more rapid, and leukocytosis is present, no time should be lost in attempting further medical methods of treatment. The abdomen should be opened and the diseased appendix removed.

Evidence of abscess, unless it is absolutely circumscribed, demands immediate incision and drainage. It is not wise to make too prolonged a search for the appendix in the abscess cavity, for by so doing the thin wall of lymph, which is nature's method of protecting the surrounding tissues from infection, may be readily broken through and a general peritoneal infection be inflicted.

If a general peritonitis is present, operative interference is still more urgently demanded, and with fair chances of success if the intestines are not already paralyzed by the infecting process. Children sometimes recover from septic (general) peritonitis. In the latter condition of affairs death is almost certain no matter what course is pursued.

If the general peritonitis is extensive, with pockets of pus here and there among the intestines, an incision should be made in the left iliac fossa as well, and the abdominal cavity mopped dry with sterile gauze. Drainage by gauze and rubber tubing is necessary, no attempt being made to close the abdominal openings by sutures.

If the diagnosis of appendicitis is certain, we believe that operation is indicated, preferably in an interval. If two or more attacks have occurred, it seems almost criminal to temporize. It goes without saying that chronic appendicitis, with all its discomforts and dangers should be regarded as a surgical disorder pure and simple.

## ACUTE PERITONITIS

This is a relatively rare affection in early life, as many of the causes originating it in adults are not operative in infancy and childhood.

**Etiology.**—Age is an important factor. In the infections of the new-born, invasion of the peritoneum through the umbilicus is not an unusual occurrence. After this period, however, the disease becomes very unusual until the middle period of childhood is reached (Holt). Sex is again important, as barring peritonitis due to appendicitis, the affection is far more common in girls.

As in the adult, peritonitis may be dependent upon bacterial invasions from other organs, by continuity, contiguity or blood and lymph channels. (Pleura, liver, stomach, perforations, intussusception, etc., through the intestine, the bladder, the female pelvic organs, the kidney, the mesenteric glands and even from the tunica vaginalis.) Most of these causes, though, are rare in infancy and childhood. Again, gall-stones, gastric ulcers, typhoidal perforations and pus tubes are not fairly common occurrences, as they are in later years.

Much has been learned recently concerning the bacteriology of these affections and we may now definitely describe pneumococcic peritonitis, streptococcic peritonitis and gonococcic peritonitis. The pneumococcus may reach the peritoneum in four ways: 1. from the pleura; 2. through the blood; 3. from the intestinal canal; 4. from the female genitalia (Clogg). Jensen's work indicates that the third pathway represents a rather common mode of invasion. Less is known concerning the streptococcic form. It may be noted when pus foci are present elsewhere (septicemia, etc.); but it may also appear as a primary affection. Gonococcic peritonitis is seen in little girls as a complication of vulvo-vaginitis.

The bacillus coli communis may also be present in acute peritonitis; but this is more commonly the case when the affection is dependent upon appendicitis. Peritonitis from this cause has been discussed.

**Pathology.**—This will depend largely upon the bacteriology. Peritonitis in the new-born and as a complication in appendicitis, we shall dismiss from this discussion. In the pneumococcic variety, we recognize pathologically, as we shall clinically, two varieties: The encysted and the diffuse forms. In both we may find pleuritis or pneumonitis, but these conditions when found are not always primary. Usually at a level below the umbilicus we find a prominence. The navel itself may protrude and pus may be seen through the navel aperture. There is mayhap an actual fistula at this point.

Where the abdominal wall is incised an abscess is found, at the level described, enclosed in a thick wall of fibrin. The intestines and pelvic organs may be matted together, and much fibrin may be deposited upon the visceral and parietal peritoneum. The pus presents the creamy, greenish appearance that every one acquainted with a pneumococcus empyema knows. Pneumococci are recovered from the fluid, from the heart's blood, spleen, and mayhap from the swollen Peyer's patches. In the diffuse form of the disease there is no attempt at walling off but fluid, exhibiting the same characteristics, fills the peritoneal cavity. Deposits of fibrin are also observed. If this form has not spread from a pleuritis, it is likely to have caused one by extension.

In the streptococcic form of peritonitis, the process is usually a general one. The pus presents a serosanguinolent appearance and there is not so much fibrin (Stoos). Streptococci are found in the fluid and probably in the blood. In rare cases "walling off" has been observed (Case reported by Stoos). As already mentioned the process may represent but a part of a septicemic or pyemic process.

Gonococcic inflammations may be localized in the neighborhood of the uterus and tubes, where we may have either an abscess formation (cul-de-sac of Douglas, pus tube, etc.) or simply a fibrinous exudate with subsequent adhesions. On the other hand, this process may become general, when we again find much fibrinous deposit throughout the peritoneal cavity and a relatively small amount of thin pus in the pelvis or lower abdomen. Examination reveals the gonococcus of Neisser.

**Symptoms.**—We shall again describe the three forms that have been accorded special attention.

The pneumococcic form starts in very abruptly, with fever, vomiting, headache and diarrhea. There is also considerable abdominal pain, particularly in the lower abdomen. With the movements there is considerable tormina and tenesmus. The abdomen is distended, tympanitic and tender, but there is little rigidity on palpation. After several days the fever may abate, but the diarrhea is likely to be persistent. After a number of days (12 to 14 according to some, 8 to 14 according to others) evidence of pus in the peritoneal cavity appears. The fever then rises again and may present a hectic type. The physical signs depend upon whether or not encapsulation and consequent localization have occurred. If so, the following signs present themselves, usually below or at the umbilicus: A local protuberance giving a pointed, rather than a well-rounded, appearance to the abdo-

men. (In contradistinction to tuberculous peritonitis with effusion.) The umbilicus may pout markedly, and if spontaneous rupture occurs, oozing of pus may actually be observed. Palpation reveals a mass and perhaps a sense of fluctuation, suggesting an ovarian cyst. Percussion also enables one to outline a map. The blood should show a leukocytosis. Dr. Stanton sent such a case to one of us, and the clear cut history, symptoms and physical signs, permitted of a prompt recognition of its nature. Dr. Harry Deaver operated two days later, finding the typical, greenish pus, fibrine formations, and adhesions.

In the diffuse forms we have a more grave illness and the signs of a generalized peritonitis. Its course is usually more rapid.

So with the *streptococcic* form we usually have evidences of a diffuse peritonitis. The child is again taken acutely ill and with much the same symptoms: fever, headache, vomiting and diarrhœa. The patient is gravely ill from the first. Tympanites appears very soon and shortly after this the signs of fluid in the abdominal cavity (movable flank dulness, etc.). The patient rapidly sinks into a typhoid state and dies. The whole course of the affection is usually not more than three or four days. Blood cultures may clear up diagnosis.

With our present day knowledge of gonorrhœa in the adult female, it is not surprising that a gonococcic peritonitis should occasionally occur in the child. Indeed, when one thinks of the frequency of vulvo-vaginitis in little girls, one is surprised that invasion of the peritoneum is not more frequent. It is a rare disease however, though a number of cases have been reported in the French, German, and American Literature. Koplik recognizes three forms:

1. The very mild ones to which the French have applied the term peritonism; 2. pelvic peritonitis with adhesions and salpingitis; 3. general peritonitis in which the child is gravely and perhaps fatally ill. Vulvo-vaginitis is nearly always observed clinically in these cases, and smears taken from it display the gonococcus. We always have cases of gonorrheal vulvo-vaginitis at the Philadelphia General Hospital, but the writers have never seen a case of gonococcic peritonitis.

**Diagnosis.**—This is difficult in the first two forms and for awhile may be impossible. In the last-named variety the gonorrheal discharge from the vagina should lead us to suspect the true nature of the affection. Abscess formation should likewise lead one to properly diagnosticate the pneumococcic form. Blood cultures alone may enable us to positively recognize the streptococcic invasion.

Acute peritonitis dependent upon one or the other of these causes must

be differentiated from that of appendicular origin. This may be hard, though diarrhea in appendicitis is very unusual. It may require an exploratory incision to settle the matter. Typhoid fever, too, is closely simulated, though blood examinations (leucopenia and the Widal test) should save us here.

With care there is little difficulty in excluding tubercular peritonitis or effusions dependent upon heart, liver or kidney diseases.

**Prognosis.**—Acute peritonitis to the student should be synonymous with grave prognosis. The gonococcus form, if localized, probably offers the best outlook, often recovering without surgical intervention. Indeed, according to some, it may do better without surgery. If more diffused, it is, or may be a grave affection, though early laparotomy may increase the patient's chances. If the pneumococcic peritonitis is localized, surgery again renders the case fairly hopeful; but the cases in which the whole peritoneum is involved are serious indeed. Streptococcic peritonitis is almost invariably fatal, though operative recovery occurred in one case where the process became localized.

**Treatment.**—The medical treatment of pneumococcic peritonitis is often unsatisfactory. Complete rest in bed should be enjoined and in the Fowler position. Cold applications (compresses of lightly filled ice-bags) may be employed for the relief of pain; though if relief does not follow soon, hot compresses should quickly replace them. Use nothing that will irritate the skin of the abdomen (turpentine, etc.) in view of operative contingencies. On account of the vomiting, it is better not to give food by the stomach, for two days at least. Some authorities depend upon nutrient enemata. These failing, employ hypodermoclysis with normal saline solution, or Ringer's solution.

The treatment for the streptococcic form of peritonitis is expressed in the single word *laparotomy*. The sooner that is done, the better. It has seemed to us that Marmorek's serum should be used freely in a condition that is so uniformly fatal.

As stated, the peritonitis dependent upon the gonococcus may do best under medical treatment. Local heat and the free use of saline purgatives sometimes accomplish wonders. The cases sometimes show unexpected changes for the worse, and the medical man should be ever on guard, and should at least demand a consultation with a surgeon under such conditions. Local abscesses and general peritonitis are certainly beyond the domain of medicine. It is a condition in which morphine or opium in some form must often be used for the relief of pain. Unfortunately it often has little effect upon the diarrhea. Hot saline enteroclysis would appear indicated, and we should feel inclined

to use an injection of nitrate of silver (1 to 2000) once daily. Bismuth salicylate or a combination of salol and bismuth subnitrate would again appear indicated. When abscess formation has occurred the patient should be operated upon. With general involvement of the peritoneum operation is usually indicated at an earlier period.

## CHRONIC NON-TUBERCULAR PERITONITIS

A few such cases have been reported by high authority; but the condition is exceedingly rare. As some authorities, equally high, still doubt its existence, we shall not enter into a discussion of it.

## TUBERCULOSIS OF THE PERITONEUM

**Etiology.**—Tubercular peritonitis is of frequent occurrence in childhood, and in the majority of cases is secondary to a primary focus, which is oftenest a tubercular enteritis, or a tubercular infection of the mesenteric glands. Munsterman, out of 2837 autopsies, and Boschke, in 226 cases of tubercular peritonitis, claim only to have discovered one and two respectively of primary tuberculosis of the peritoneum. The lymph-channels have been proved the common carriers for the transportation of the bacilli from some distant or near focus, such as a tubercular intestinal ulcer or a caseous degeneration of a mesenteric gland. The genital tract of the female occasionally offers a mode of infection, as is illustrated by the frequently quoted case of Vierrordt: A girl six and a half years old developed tubercular peritonitis while suffering from vaginal discharge in which the tubercle bacillus was found. A good recovery was made after eliminating the vaginal focus of infection. R. Abbe calls attention to the probability of milk food acting as a carrier of the bacillus, and suggests possible penetration of the intestinal follicles by the bacilli as the most reasonable method for their entrance in certain cases in which no other invading focus can be found. Such a path of invasion is probably rare, however.

**Pathology.**—Tuberculosis of the peritoneum may be but a part of a general miliary infection which, according to Zeigler, is evidenced merely by gray translucent tubercles of small size dotting the entire surface of the peritoneum, but without extensive inflammation. There may be some slight injection of the membrane, the latter being generally transparent, smooth, and glistening. In the form under discussion, in which the tuberculous process predominates in the

peritoneum, inflammatory changes take place, leading to the formation of a liquid exudate. The peritoneum is thickened and opaque, connective tissue being quickly developed, causing this density. Adhesions of the intestines, numerous tubercles, and caseous deposits are to be found embedded in the infiltrated peritoneum. These are often concealed by the newly formed fibrous tissues. The exudate, which varies generally in quantity, may be composed entirely of serum, or, on the other hand, may contain fibrin, pus-cells, and occasionally blood-



FIG. 32.—CASE OF TUBERCULOUS PERITONITIS.—(From D. G. M. Wells, Portland, Oregon.)

corpuscles. Perforation of the intestines or abdominal wall may take place. The process ends in either absorption of the inflammatory exudate with caseous metamorphosis of the débris, or the infective process may appear in other organs and end life. In other cases the process may be more or less local and one may find an encysted exudate. In still a third group of cases, the greatly thickened layers of the peritoneum may be bound together most firmly, as are the intestines. There is no demonstrable fluid exudate. The omentum may be rolled up and simulate an abdominal tumor (sarcoma of the kidney,

etc.). One of us saw such a case, in which even the roentgenogram appeared to verify the clinical diagnosis. At the operation only the condition described was found (see Abdominal Tumors).

**Symptoms.**—The symptoms will depend upon the character of the pathologic process existing in the peritoneum. If the disease is but a part of a general miliary tuberculosis, the attack is of sudden onset and the symptoms are those of acute general peritonitis, except when, after a short intermission of symptoms, a fresh outburst occurs. The fever, which is generally of true hectic character, may, after a time, completely subside, but following this reduction of temperature there is no improvement in the general health. Emaciation becomes marked, and from the coalescence and adhesions of the intestines emesis and obstinate constipation supervene. The infection being general, death usually results

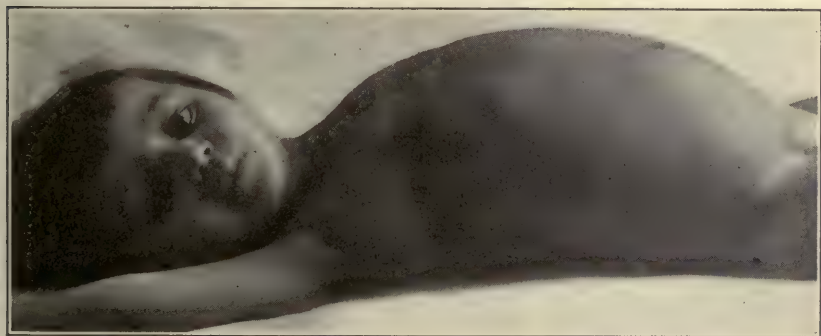


FIG. 33.—TUBERCULOUS PERITONITIS IN A THREE-YEAR-OLD CHILD.  
Note the dilated veins.

from the more acute process in the lungs. On the other hand, when the peritoneum has become infected from a mesenteric gland, or an intestinal ulcer, or some more remote tuberculous focus, the picture presents a slow, wasting disease almost identical in nature with chronic peritonitis from other causes, differing, however, in the fact that the febrile symptoms, which at first are hardly perceptible, soon become prominent, manifesting themselves by slight exacerbations of the temperature in the evening. Accompanying this rise of temperature there are generally night-sweats. The pulse is usually weak and small, and as in other tuberculous conditions, its rate is disproportionately rapid when compared with the temperature. The physical signs are usually prominent and distinctive, particularly when free fluid is present in the abdominal cavity. Then the abdomen is very prominent and the superficial veins are dilated. The umbilicus too often presents the characteristic ("pouting") prominence. Palpation often elicits

tenderness, increased density and fluctuation. The latter sign is best determined by tapping or flipping in one flank while the other hand is placed on the opposite one. Another person should place a hand firmly in the median line of the patient's abdomen, so as to interrupt waves passing over the abdominal wall. As mentioned, palpation may apparently reveal a solid tumor in the plastic cases. Percussion reveals flank dulness and dulness over the lower abdomen, and unless the amount of fluid is too large, a circular area of tympany in the neighborhood of the umbilicus. These percussion finds are secured with the patient in the supine position. If he is caused to rest on one or the other side, and the fluid is movable, tympany appears in the flank



FIG. 34.—TUBERCULOUS PERITONITIS.

that is elevated (movable dulness). Resistance to the pleximeter in percussion is much increased. Plastic cases may be hard to diagnose. Diarrhea may be present, when there is tubercular enteritis. The little patient is peevish and much emaciated. The thoracic organs may be free from infection, though a careful clinical search will often reveal a primary focus, and not seldom some apical involvement. Death results from hemorrhage, occlusion of the intestines, strangulation, marasmus, general tuberculosis, or from exhaustion.

**Diagnosis.**—This is based upon the history, the general symptomatology (particularly the temperature, pulse, wasting and hectic) and the physical signs described. The ophthalmic and cutaneous reactions (Calmette, von Pirquet and Moro) may aid us, though only positive reactions should be viewed as evidential. Tubercle bacilli may also be found in the stools. The disease should be distinguished from peritonitis dependent upon other organisms, from ascites of other origin and from abdominal tumors.

**Prognosis.**—Until very recent times when treatment has accomplished so much for tuberculous subjects, the outlook in this disease has been viewed as bad. Both of us have seen a patient with a well-marked tuberculous peritonitis recover. The day had been set for operation in this case; but the temperature and the effusion commenced to subside. The prognosis depends largely upon the extent to which other organs are involved. It has been claimed that surgery accomplishes much more for these patients than medicine, but some recent statistics have thrown doubt upon this commonly accepted view. From the operative standpoint the cases with effusion are the most favorable.

**Treatment.**—These patients should be at perfect rest. They should remain in bed as long as the temperature shows the tendency toward evening rise. The rest should be secured in the open air, however. Forced feeding should also be employed, though the diet may have to be modified considerably when diarrhea (often from tuberculous enteritis) is present. We employ tuberculin in these cases,—beginning with small doses and following the method of Trudeau. If the patients who have considerable effusions do not improve under this treatment, simple incision and drainage should be advised. Even then, operation is contraindicated, when extensive tuberculous lesions exist elsewhere. In plastic cases operative measures are usually unsuccessful.

## CHAPTER VII

### DISEASES OF THE LIVER

Diseases of the liver are comparatively rare in childhood, and have received singularly little attention at the hands of systematic writers on pediatrics. Disorders of the liver are more frequently due to secondary changes, usually preventable, than those of almost any other organ. The causal processes of these operate over long periods of time, and it is a quality of infantile tissue to escape many of them. Moreover, this very rarity limits the possibility of any one observer coming in contact with many instances of organic hepatic disease, rendering them less ready to invite much literary attention. In consequence of this, students are not well instructed, and instances of liver disease, when they do occur, receive less recognition and attention than their importance warrants. Functional disorders of the liver occurring in late childhood and early adolescence, however, we are told by Musser, are probably of much more frequent occurrence than we are led to believe from the text-books. Before taking up the subject of the various diseases separately, a few remarks as to the general symptomatology of affections of the liver are in order.

**General Symptoms.**—In a general way the symptoms of hepatic disease in childhood are the same as in the adult; thus we have the phenomena pointing to general failure of health and strength; there is a loss of appetite and other dyspeptic symptoms. The bowels are either constipated or irregularly loose. If there is obstruction of the common duct or of the hepatic duct the stools may be clay colored (acholic) and very offensive. More or less jaundice usually accompanies the condition. In certain diseases of this class ascites is a prominent feature. Enlargement of the spleen and of the veins of the abdomen are also occasionally seen. From the congestion affecting the lower bowel hemorrhoids are frequently produced. In certain affections, particularly those accompanied by formation of pus in the liver, chills, fever, and sweats may occur. Various nervous disturbances, such as intense mental depression, amounting sometimes to an actual melancholia, are nearly always present. Pain is only present in certain acute diseases, such as suppurative hepatitis, in acute congestion, and in syphilitic disease of the organ, especially

where the capsule is involved. When present, it will be localized or general, rather more generally the former. It is felt on the right side of the hepatic region, and may extend to the right shoulder. As a general rule, it is dull and heavy in character, although in certain forms of hepatitis it is sharp and cutting, very closely simulating that of right-sided pleurisy or pleurodynia. However, we know that in pleurodynia we have immobility, the respirations and general movements are painful, the parts affected being tender when palpated or subjected to other methods of examination. As a general rule, also, there may be rheumatism in other parts of the body. In pleuritis the pain is distinctly increased during respiration, and is always associated with pleural friction sound. Besides this, a cough of characteristic type always attends the attack. Occasionally the symptoms of perihepatitis may so simulate those of pleurisy affecting the right side that it is almost impossible to tell them apart, but in pleurisy, sooner or later, fluid will be detected in the cavity of the pleura, while in hepatitis, of course, no such symptom will develop. The pain may also be distinctly paroxysmal, as in hepatic colic, but this affection is found so rarely in young children that it may almost be left out of the question. Pain from liver disease is apt to be constant, and is most usually increased by movement or pressure. It is liable to extend upward along the lower edge of the organ or into the epigastrium. In hepatic abscess the pain is localized, and where the abscess has resulted from traumatism, the position of pain corresponds to the point of injury. In disease accompanied by enlargement of the organ sensations of heaviness and weight are sometimes experienced on the right side.

During the early years of life the liver, in proportion to the bodily weight, is much larger than in the adult. The upper border extends to the fifth, sixth, and seventh ribs in the midclavicular, axillary, and scapular lines respectively. The lower border can be outlined two centimeters (more or less) below the margins of the ribs in the right midclavicular line. The left lobe can be outlined with considerable ease, and extends in the median line to within an inch of the umbilicus.

In order to make a systematic examination of the organ the methods to be employed may be divided into inspection, palpation, and percussion.

**Inspection.**—In inspecting the abdomen and thorax of a patient suffering from hepatic disease little information can often be obtained, except in certain forms of disease. The abdomen may be somewhat distended, sometimes by flatulence and, in certain affections, by ascites. If much enlargement of the liver is present, the lower third of the right

side of the thorax and the upper part of the right side of the abdomen may be unduly prominent, and the breathing correspondingly affected on that side. Especially is the latter the case if much pain is present. In abscess and hydatid disease tumors may be outlined in the left lobe and along the lower border of the right lobe. Occasionally in abscess the skin above the affected area of the liver may assume a red color. Enlargement of the veins of the abdomen will very frequently be noticed, especially in certain diseases. The position assumed by the patient is of some value in diagnosis. When lying down, the patient will usually assume a posture on the right side, with legs drawn up; turning on the left side increases the amount of pain considerably. The breathing will often be shallow, on account of the pain felt in prolonged inspiration. The presence or absence of jaundice should be noticed.

**Palpation.**—The large size of the left lobe of the liver may sometimes be mistaken for a tumor, either on the surface of the liver or in the abdominal cavity. The latter may be diagnosticated by the fact that the liver will move during respiration, whereas a tumor would remain stationary. The normal outline and consistence of the liver may be changed in various forms of disease affecting it; thus, in amyloid disease a distinct induration of the surface and edge may be detected by palpation, and where fatty infiltration has taken place, these parts will be found to be smooth and soft, while in cirrhosis they are sharp and hard. A characteristic fremitus will very often be detected in hydatid disease, and a friction sound in perihepatitis. In palpating and in percussing the lower borders of the liver care should be taken to empty the transverse colon thoroughly previous to examination, as a quantity of feces contained in this part of the bowel will seriously interfere with the examination. We prefer the Edebohls method of palpation. (See Physical Examination.)

**Percussion.**—In the practice of this method of diagnosis it should be remembered that in order to outline the upper borders of the liver deep percussion must be employed, while it is best to use light percussion in defining the lower border. These rules only apply to older children, however, as they do to adults. In pursuing percussion studies in infancy and early childhood, light percussion should always be employed. One should also depend as much upon the sense of resistance offered to the pleximeter as upon the percussion note. By means of percussion variations in the size of the liver may be detected. An irregular enlargement of the organ may point to a hepatic abscess or hydatid disease. In both of these the increase in dullness is over

the convexity, if this part of the organ is affected, the area running upward and to the right; when the center of the organ is affected, the line of dullness is downward. When the left lobe is enlarged, an increase in size of this portion can be quite distinctly outlined. When fluid is present, the area of dullness may be made to change by turning the patient on the left side. A tumor of the right kidney may in some cases give the sensation of being attached to the liver or may simulate enlargement of that organ, but a line of tympany or a lower note will be detected, and the finger will slip between the lower borders of the liver and the tumor. When doubt as to the diagnosis exists, the history of the patient and an examination of the urine will generally settle the question. Exploration of the liver can also be aided by aspiration with a hypodermic syringe. By this method specimens of pus, serum, or hydatid fluid may be drawn off and examined. Pus from an abscess in the liver will contain, principally, leucin and tyrosin, and possibly the characteristic liver-cells. When the fluid is reddish-brown and mixed with blood, and especially when the *amœbæ dysenteriae* are found in it, the abscess is secondary to dysentery. When serum is drawn by the hypodermic needle, we may conclude that it comes from the pleural cavity and not from the liver, as the latter does not yield serous fluid. The fluid from a hydatid is of low specific gravity, alkaline in reaction, and clear. It contains small quantities of albumin, sugar, and a considerable amount of sodium chlorid. Succinic acid may also be found. The echinococcus-membrane, hooklets, and other traces of the parasitic cause of the disease will be found on microscopic examination.

Congenital stenoses and atresias of the biliary passages have been mentioned in an earlier section (Icterus in the new-born).

### CATARRHAL JAUNDICE OCCURRING DURING CHILDHOOD

**Etiology.**—It is probable that most cases of so-called catarrhal jaundice are of infectious origin. Indeed, epidemicity has been so marked at times that certain authorities use the term infectious jaundice. The jaundice of very early life is certainly of this nature in most cases. Again, jaundice sometimes appears in certain of the infectious diseases. We have seen it several times in epidemic influenza, and in scarlet fever it is considered a very ominous sign. In one case of Weil's Disease, Jæger found the *bacillus proteus fluorescens* in the urine. Food does seem to play an etiologic rôle, however, as some cases reported from Saxony seemed to prove. A father

prescribed the so-called "honey-cure" for his children on two occasions, and both times they were seized by attacks of jaundice. Atmospheric causes and taking cold also seem to be important, probably as predisposing causes. Jaundice has resulted from large doses of male fern, santonin, lactophenin and tuberculin (Stoos).

**Symptomatology.**—Jaundice may occur at any time in childhood, although the period immediately following birth is the one in which it is most frequently seen. The symptoms are usually of gradual onset, being those of a more or less severe gastric or intestinal catarrh, either of acute or subacute origin. The liver is found to be enlarged upon percussion, its outline extending below the normal line for an inch or two. There is generally, also, some tenderness in the epigastrium and right hypochondriac region. With these symptoms there is also the typical coloring of the skin, which may vary from a yellow to a brownish-yellow or almost greenish tint. The ocular conjunctiva, and even the mucous membranes of the body, may share in the general discoloration. The urine is also discolored and Heller's test exhibits the classic play of colors. The bowels are constipated and present the typical clay color and offensive odor. Occasionally a slight rise of temperature accompanies these symptoms. An itching of the skin, which may be quite distressing, is frequently felt. Riesman has described a preicteric heart murmur.

The **diagnosis** is easy; the discoloration of the skin, the slight pyrexia, the history of indiscretions in diet or chilling of the extremities should materially aid in distinguishing the condition. It may be that the case is observed in an epidemic, such as have occurred so often in Switzerland.

**Prognosis.**—The prognosis is usually good, though severe cases may occur. Catarrhal jaundice rarely occurs twice in the same subject (another evidence of its infectious nature).

**Treatment.**—If the attack of jaundice is severe, particularly if fever accompanies it, the patient should be kept in bed for a few days. Mild counterirritation may be made over the epigastrium by rubbing the parts with some stimulating liniment or by the application of a mustard or spice bath. Massage is of considerable use, particularly where manipulations can be made over the gall-bladder. Faradic electricity has also been advised. The diet is a matter of very great importance. No foods containing starch or sugar should be employed nor should fatty foods be given the patient. The patient should be kept upon a diet of milk, diluted and made alkaline by lime-water or some alkaline mineral water. Musser advises that this milk be taken

hot. Animal broths, particularly mutton broth, thin chicken broth, or beef-tea, should be the principal food employed. When the digestion is weak, particularly if the patient is inclined to vomit, oyster or clam broth or koumiss may be used with advantage. As the patient improves he may be given small quantities of fresh fish or eggs and the white meat of chicken. The medicinal treatment is of considerable importance in jaundice. If seen early, very frequently a brisk laxative of calomel, combined with phosphate of soda, may be given two or three times in the twenty-four hours, this being followed by a moderate dose, say a dram, of castor oil. The combination of calomel and bismuth has also been found to be of use. The following formula has been recommended by Musser:

R. Liquor potassii citratis..... fl. ʒij  
 Tinct. opii camph..... fl. ʒj.  
 Sig.—One-half to one teaspoonful every two or three hours.

This formula is particularly useful when there is considerable pain. When vomiting is the prominent symptom, minute doses of hydrochlorate of cocain are of value. In the treatment of this condition the diet should be such as will be quickly assimilated, with the formation of as little gas as possible. For the medicinal treatment, extract of pancreatin in combination with an alkali, given an hour or so after meals, is the most valuable agent. Napthalene, salol, thymol, creasote, and particularly charcoal, may all be used with good effect in certain cases. As a combination of the two last-named agents, Musser recommends the following:

R. Creosotum..... gr. 1/4  
 Carbo. lig..... gr. j  
 Pancreatin..... gr. j  
 Bismuth. subnitrates..... gr. ii j  
 Sig.—Ft. chart No. i. Take after meals.

To relieve the distressing itching of the skin, which is sometimes so severe as to cause almost continual scratching, a sponging of the surface with ten drops of carbolic acid to a pint of water, or with a hot solution of borax or bicarbonate of soda, will be found very useful. If this fails, Goodhart recommends that from 1/32 to 1/24 of a grain of pilocarpin should be injected hypodermically. For the cerebral symptoms, in severe cases, effort should be made to hasten as much as possible the elimination of bile, at the same time using all means possible to support the patient. For this purpose the salts of ammonia, particularly the chlorid, should be given in doses of from one to five grains, and administered in the syrup of orange or syrup of licorice, or, better, a

little glycerin. Phosphate of soda is also probably one of the most valuable drugs that can be employed to hasten the elimination of bile. Caffein has also been extensively used, and pilocarpin is a valuable agent in aiding diaphoresis. When the temperature becomes subnormal, and especially if this is accompanied by considerable prostration, the patient should be fed on a nutritious, highly concentrated diet of proteid foods, and stimulation aided by moderate doses of alcohol. When hemorrhages occur, sulphuric acid and the acetate of lead are useful as astringents. Turpentine and ergot have also been employed for this purpose. When the blood is more than usually depleted by the disease, the use of oxygen has been recommended. As the patient improves he may gradually return to a more varied diet, although for a long time fatty, saccharine, or starchy foods must be used with great caution. As an aid to digestion and also to stimulate hepatic action, probably one of the most useful medicinal agents is hydrochloric or nitrohydrochloric acid. These may be given internally in doses of one or two drops, combined with some bitter tonic. Local applications of the diluted acid, applied in the form of a wet-pack over the hepatic region, have been used with very good results. Another method which has been recommended by Musser, and which was invented by Krull, is the injection of from two to four pints of water into the colon three times a day. The temperature of the water is raised with each injection; at the first a temperature of 59° F. (15° C.) is used, and the other two enemata are made warmer until a temperature of 72° F. (22.2° C.) is reached.

## CONGESTION OF THE LIVER

**Etiology.**—Two forms of hepatic congestion are described—namely, the active and passive. The former is produced by an exaggeration of the normal congestion of the organ, which is produced by the stimulus of food. The causes of this increase in blood supply are generally overeating, the food being either too rich in quality or of a too great quantity; an abuse of stimulants may also produce the condition, but this is rare in childhood.

The passive form of congestion in almost every case occurs as a secondary consequence to diseases of the heart or lungs. The liver becomes engorged with blood, this condition being due to the deficient action of the lungs or heart. Chronic malarial poisoning is also given as a cause.

**Symptoms.**—Some pain is experienced in the region of the liver,

and the organ will be found to be enlarged and tender on palpation. The increase in size will be uniform, sometimes extending for a distance of one or two inches beyond its normal boundaries. The edges and surfaces are smooth, no nodules being felt. When the gall-bladder is enlarged, it is possible to outline it in the right hypochondriac region to the left of the midclavicular line, in a line drawn from the acromion process of the right scapula to the umbilicus (Musser). The amount of jaundice is usually slight. The increase in size is slow, constant, and uniform. According to Musser, the edge of the liver is sharper in the passive than in the active variety of congestion and is more indurated. "In the right midclavicular line the lower border may extend so the level of the umbilicus, and in the median line the left lobe may extend for three-fourths of that distance." Where there is effusion in the right pleura, the upper border cannot so easily be made out. The constitutional symptoms are those of gastro-intestinal catarrh: there is loss of appetite, some nausea and vomiting, constipation, and intestinal dyspepsia. The tongue is covered with a brownish coat; the amount of jaundice is usually slight (sub-icteric). In the passive form we have, in addition, the constitutional symptoms of organic diseases of the heart, lungs, or kidneys, affections of these organs being the most frequent cause of passive congestion of the liver. The urine is apt to be albuminous and contains considerable quantities of bile pigments. A moderate amount of mental depression very frequently accompanies the disease.

The **prognosis** of the acute form of congestion is favorable. In the passive variety the prognosis will be influenced by the extent and progress of the disease causing the congestion.

**Treatment.**—The main objects to be accomplished in treatment are the removal of the cause of the congestion and the relief of the engorged liver by the judicious use of purgatives. Diseases of the heart and malaria should receive particular attention as causes of the passive congestion, for appropriate treatment may rapidly clear up the hepatic condition. The patient should be put on a low diet, and all starchy, saccharine, and fatty foods excluded. The diet should consist of animal broths, or small quantities of meat and such other foods as are digested chiefly in the stomach. Purgatives are of great use, particularly those which act directly on the liver. The two most useful of this class of agents are calomel and phosphate of soda. These may be given either alone or in combination. When combined, they are best administered in the form of a powder, capsule, or combined with the *mistura glycyrrhiza*. Phosphate of soda is

best administered at bedtime or in the morning. It may be given in hot water, soup, or broth. Chlorid of ammonia, in doses of from three to five grains every two or three hours, has been greatly praised. Ipecacuanha has also been used with great benefit. It, however, sometimes produces such an amount of nausea and depression as to reduce its favorable action. According to some authorities, it should be administered in doses as large as five grains, twice in the twenty-four hours, to children under five years of age. A few drops of deodorized tincture of opium or a sinapism over the epigastrium are used to prevent the intense nausea. After the acute symptoms have subsided, dilute nitric acid or nitrohydrochloric acid may be given in doses of from two to ten drops three or four times a day, or local applications in the form of packs, consisting of cloths wet with diluted nitric acid, may be applied over the region of the liver. The dyspeptic symptoms should be treated by the use of bitter tonics and continued small doses of calomel, bismuth, or phosphate of soda. Small doses of silver nitrate may also be used with benefit. The passive form is best relieved by treating the condition causing it.

### HEPATIC ABSCESS

**Synonyms.**—Suppurative Hepatitis; Suppurative Inflammation of the liver.

**Etiology.**—This condition, as has been before stated, arises usually from infection somewhere in the portal area; thus, injuries or disease of other abdominal viscera may produce it. Appendicitis is a very frequent cause. We saw the specimens from a case (Willard and Jump) in which a pin was present in the vermiform appendix, and the liver contained many small foci of suppuration. In tropical countries amebic dysentery may be followed by abscess of the liver. Here the abscess is usually single. Trauma is another important cause. Round worms (as already stated) have wandered into the biliary passages and abscesses have resulted. Tuberculosis is another rare cause, as are also typhoid fever, phlebitis, etc. Legrand (quoted by Stoos) collected 112 cases from the literature. Of these 31 were dysenteric (mostly tropical), 19 traumatic, 15 appendicular, 10 tuberculous, 13 due to worms, 66 typhoid fever, 2 phlebitis of the umbilical vein, and 6 of doubtful origin.

**Pathology.**—As stated, several times the abscesses may be single or multiple. The single ones are usually caused by direct traumata or by amebic dysentery. In the latter case ameba are recovered from the

thick reddish pus. The whole liver is enlarged and congested and perihepatitis may exist to a considerable degree.

**Symptoms.**—Usually the symptoms of multiple abscess of the liver are preceded by indications of disease in some other abdominal organ. The special symptoms of abscess are: Jaundice suddenly appears (fifty per cent. of cases), or, where this is not very marked, the skin assumes an unhealthy, sallow hue. The liver becomes enlarged and painful, this pain being of a heavy, dragging character. Fever of an intermittent type is present, and there are daily rigors. In some cases the fever somewhat resembles typhoid. The tongue becomes dry and covered with a brownish coat. There are sordes on the teeth and lips. Nausea and vomiting are present and are accompanied by diarrhea, the stools being light colored and offensive. The urine rapidly diminishes in quantity, is highly colored, and contains much bile pigment. Albumin is present. A microscopic examination of the urine will demonstrate the presence of blood and granular and epithelial casts. The nervous system soon becomes involved, delirium of a low, muttering type appearing. Subsultus is present. Later the patient may have convulsions or may pass into a state of coma. Death may occur from exhaustion, or in some cases the kidneys become so involved that nephritis may produce a fatal termination. In addition to the enlarged liver, one may palpate the local swelling or swellings upon its surface or may detect actual fluctuation.

**Diagnosis.**—In acute single hepatic abscess resulting from traumatism or ameba coli the diagnosis can be made by the history of injury or dysentery, the irregular enlargement of the liver, and the symptoms pointing to suppuration. In doubtful cases the exploratory needle may be inserted, under strict antiseptic precaution, to aid in the diagnosis.

When multiple abscesses exist, the symptoms of hepatic abscess following evidences of disease in some other abdominal viscus should lead us to suspect the presence of this form of hepatic inflammation.

**Prognosis.**—When the abscess is single and can be opened externally, a favorable termination of the case is possible. The prognosis of multiple abscess is very grave.

**Treatment.**—The treatment should be by surgical methods. According to the best authorities, where the number of abscesses does not exceed three, free incision should be made. When the abscess is situated along the margin of the ribs or is in the epigastric region, the operation is simple. When situated in the convexity of the right lobe,

Musser advises that it should be opened through the pleural cavity, and in this case excision of the ribs is necessary. The abscess cavity should be drained and irrigated, and a drainage-tube inserted.

## CIRRHOSIS OF THE LIVER

We must recognize five varieties of cirrhosis in early life, though none is common; viz.: 1. Atrophic alcoholic cirrhosis (hobnail liver, gin drinker's liver, etc.); 2. hypertrophic cirrhosis (Hanot's disease); 3. cirrhosis due to circulatory disturbances; 4. cirrhosis from congenital obliteration of the bile ducts; 5. syphilitic cirrhosis.

**Etiology.**—Most of the cases of true gin drinker's liver have been reported from England, where the custom of giving spirits to babies is not unusual among the lower classes. Cases so arising have also been reported from France and Germany. This history is not obtainable in all cases of atrophic cirrhosis, however. It has been observed in the syphilitic (though this will receive attention in another section), in the tuberculous and after certain infections.

Some authorities have claimed that atrophic cirrhosis is more common than the hypertrophic form; but Stoos states just the reverse. He says that Hanot's disease is the form more frequently observed in childhood. The cause of hypertrophic cirrhosis is not known, though Hutinel believes it to be a subacute infectious hepatitis. The cirrhosis caused by circulatory disturbances may be said to be due to adherent pericarditis dependent upon rheumatic or tuberculous cause. Congenital obliteration of the bile ducts is frequently accompanied by a considerable degree of sclerosis. Whether the obstruction is responsible for the sclerosis or not is a moot question. That syphilis causes the last-named variety is of course clear. Cirrhosis occurs far more frequently in male than in female children.

**Pathology.**—The pathology of the atrophic form does not differ from that of the adult disease and need not be dealt with here. In the hypertrophic form, the liver may be but little enlarged, while the spleen is very large. Ascites is not so likely to be present as in the atrophic form. Joint deformities (particularly of the smaller joints) may be present. The third form of cirrhosis as already mentioned is found associated with adherent pericarditis, pleuritis, and peritonitis (polyserositis). Tubercular deposits may be found (cardio-tubercular). This form has been described under various titles—pseudo-cirrhosis due to pericarditis (Fick), the sugar cake liver (Curschmann), etc. In cirrhosis due to congenital obliteration of the bile ducts we may find

either stenosis or atresia of the hepatic, cystic or common bile duct. The gall-bladder may be enormously distended on the one hand or absent on the other. Koplik describes four forms of syphilitic disease of the liver, viz.: a. The form in which gummata are found (this has actually been observed at birth (Fournier)); b. the diffusely cirrhotic form; c. the lobulated liver in which connective tissue divides the organ into sections; d. the so-called miliary syphilis of the liver. Collections of round cells are found much resembling tubercles.

**Symptoms.**—In the beginning the symptoms of atrophic cirrhosis are very apt to be confounded with those of ordinary hepatic congestion arising from disturbances of the digestive tract. There are nausea and vomiting or attempts to vomit, these occurring particularly in the morning. The vomited matter consists largely of mucus. The bowels are irregular, being sometimes constipated, while at others attacks of diarrhea may occur. The stools contain considerable mucus. Hemorrhages may take place from the nose, mouth, esophagus, stomach, or intestines, and purpuric spots are occasionally seen in different parts of the body. The face is pale or of a sallow hue, and numerous stigmata, composed of groups of minute veins, are seen upon it. There is a slight amount of jaundice in about one-half of the cases. This may be constant, or attacks may simply recur at intervals. A moderate amount of ascites is almost invariably seen, and not infrequently this may be one of the prominent symptoms of the disease. From obstruction to the portal system there results dilatation of veins, large and small, particularly the superficial veins of the thorax or abdomen. Not infrequently a well-defined arch is seen extending across the chest, marking the attachment of the diaphragm. Enlargement of the spleen always occurs. The temperature in the early stages is moderately elevated, a rise of two or three degrees being usual— $101^{\circ}$  to  $102^{\circ}$  F. ( $38.3^{\circ}$  to  $38.9^{\circ}$  C.). This is most commonly observed in the evening. As the disease progresses, however, a subnormal temperature is the most common. The urine is of high specific gravity and contains large quantities of uric acid and urates. If nephritis develops, which it very frequently does during the course of the disease, the urine will become albuminous and will be found, on microscopic examination, to contain granular and hyaline casts. Traces of sugar will sometimes be detected. During the first stage the liver enlarges, but later steadily diminishes in size, this decrease being particularly noticeable in the left lobe. While the dropsy usually seen in this disease is abdominal, yet in a certain number of cases swelling of the lower extremities may take place. In the late stages of the disease the mind becomes

clouded, largely from retention of the various products of excretion, the patient becoming first dull, and later passing into coma or active delirium. Convulsions may occur.

*Hanot's Disease* (hypertrophic cirrhosis) is usually a slow affection, the patient living for several years. In infants its course may be more rapid, however. The children affected by it are stunted in growth and are intensely jaundiced. Their extremities seem very small compared to their trunks. The liver is found enlarged and firm to palpation, and the spleen usually more so. Ascites, if it develops at all, comes late.

The circulatory form is recognized by its physical signs of adherent pericardium, pleural effusion, enlarged liver and peritoneal effusion. There may also be a history of rheumatism or clinical evidence of tuberculous disease. Congenital obliteration of the bile ducts usually manifests itself soon after birth in obstinate jaundice, acholic stools, atrophy, enlargement of the liver and possibly of the gall-bladder (fluctuating mass). Death may terminate in hemorrhage, particularly from the navel.

**Diagnosis.**—The diagnosis is to be based upon the physical signs, the symptoms of portal obstruction, and the characteristic appearance of the face. The diseases with which cirrhosis is most likely to be confounded are, first, atrophy of the liver; but cirrhosis most commonly follows obstructive diseases of the heart or lungs. In atrophy no nodulation of the organ occurs, and the history of alcoholism or syphilis is generally wanting. Second, cirrhosis must be differentiated from tubercular peritonitis: In this disease the abdominal tenderness is general; there is rapidly developing ascites, absence of jaundice, and, in the majority of cases, the symptoms of gastro-intestinal dyspepsia are absent. On percussion the liver will be found to be normal. There are also, as a general rule, evidences of tuberculosis in other organs of the body. Cancer of the peritoneum may have some symptoms resembling those of perihepatitis, but this disease occurs very rarely in childhood. The liver will be found normal upon examination, and there will be other symptoms characteristic of cancerous disease.

The diagnosis of syphilitic cirrhosis is based not only upon the physical signs obtained in a clinical study of the liver, but also upon the history and the other lesions of inherited syphilitic disease.

The **prognosis** is very unfavorable in practically all forms except the syphilitic. Here early treatment may meet with gratifying results.

**Treatment.**—The indications for treatment are, first, to remove the causes; second, to prevent, so far as possible, the increase of connective-tissue growth, and to relieve the engorgement of the hepatic and portal circulation. Third, if the case is too far advanced for cure, we must then endeavor to relieve the symptoms as they arise. In order to meet the first indication the causes of irritation must be removed. The patient should be allowed no alcohol in any form; all highly seasoned, fatty, or saccharine foods must be given up, and even articles of food containing starch must be used with great caution. The diet should consist principally of the lighter meats, such as the white meat of chicken or turkey, although other animal diet may be employed in moderation. To better insure its digestion it is well to have the meat finely chopped, and made into the form of a meat ball, without fat, and carefully browned. As a continuous animal diet may tend to cause scorbutus, doses of lemon-juice or diluted citric acid must be administered occasionally to counteract this tendency. Eggs may also be employed as an article of diet.

Probably the best food for these cases is milk—either the best dairy milk, skimmed, or buttermilk may be used—or in some cases where the patient tires of this article of food, which is generally the case in a short time, it may be administered in the form of various desserts, such as junket, blanc mange, or light puddings. The tendency to constipation must be corrected by the administration of alkaline aperient waters, such as Hunyadi, Saratoga, Carlsbad or Vichy. Vegetables such as spinach, lettuce, or those containing little or no starch may be allowed, but potatoes, rice, and other vegetables containing much starch should be used sparingly, if at all. The question of dress is of some importance. The patient should wear flannel next to the skin the year round, and should dress warmly enough to avoid chilling. An outdoor life in the open air is best for these cases. The medicinal treatment should be directed toward the relief of the engorgement and the prevention of the increase of the pathologic conditions causing it. The application of cups and leeches, which has been practised by some, is not now recommended by best authorities, although counterirritation over the liver by the use of some stimulating liniment may probably be of some slight use. The benefit derived by having the patient drink freely of hot water, say a glassful half an hour before eating, each draft to contain from two to five grains of sodium phosphate, is very considerable. The saline cathartics or laxative mineral waters are exceedingly beneficial. The patient should have from three to six liquid movements a day. To prevent the increase of

connective tissue in the organ many drugs have been recommended. One of the most highly lauded is chlorid of ammonia. This should be given in doses of from two to five grains every four or five hours. It is best administered in some syrup or elixir. Probably one of the most useful agents which has stood the test of time is phosphate of soda; this should be given in doses of from five to ten grains, according to the age of the child. It is best administered in hot water, and may sometimes be disguised in soup, being used in the place of salt. Potassium iodid is said to retard the changes in the organ when given in the early stages. In cases of syphilitic origin this drug, of course, will have a particular usefulness. Small and continued doses of mercury, in the form of calomel, bichlorid, or gray powder, are valuable agents at all stages of the syphilitic infection. In advanced cases, where the principal indication is the relief of the various distressing symptoms of the disease, these should be treated as they arise. Where ascites is marked, the patient should be placed on a dry diet, principally of meat. The gastro-intestinal indigestion which always accompanies the malady should be treated as in other cases. Hemorrhage from the stomach or other part of the digestive tract requires rest in bed, the administration of cracked ice, and the application of an ice-bag externally. Where the hemorrhage is of gastric origin, food should be administered by rectum. Opium should always be given to quiet the patient, and may be used in either the form of morphin hypodermically or paregoric in suitable doses, administered alone or in combination with an astringent, such as sulphuric acid. The following formula, recommended by Musser, is useful in these cases:

R. Tinct. opii camph.

Acid. sulphuric. aromat. . . . . aa fl. ʒj.

Sig.—Eight to ten drops in water every two, three, or four hours.

Various astringents, such as acetate of lead, bismuth, nitrate of silver, and many others, have been employed. Hamamelis, in doses of twenty drops every hour or two, has been employed with benefit. The various preparations of iron, particularly Monsel's salt given in the form of a hard pill of one grain, is of use in intestinal hemorrhage. In cases of hemorrhage from the lower bowel, astringent enemata should be employed. Ascites should be treated by the administration of diuretics and saline cathartics. Drafts of cream of tartar dissolved in water are valuable for this purpose, and are rather pleasant to the patient. Infusion of scoparius has also given good results in these cases, but probably the best of all drugs for this purpose is calomel.

This may be used alone, or in combination with a compound jalap powder. Copaiba gives a more permanent result. Where the child is old enough to take the drug in this form, it may be administered in the capsule containing three minims, to be taken every four hours. Where the heart is weak, caffein or digitalis should be employed, and hydrochlorate of cocain is also said to have given good results for this purpose. The use of large doses of glonoin, testing it carefully in the individual case, sometimes accomplishes remarkable results. This treatment was suggested to one of us by the late D. E. Hughes. As in the late stages of the disease death frequently takes place from exhaustion, stimulants and cardiac tonics should be used where this condition is threatened. Paracentesis may be employed frequently. Palma's operation was devised to open up new pathways for the portal circulation. In a disease so desperate as atrophic cirrhosis it seems worthy of trial.

### FATTY LIVER

The so-called fatty liver consists in a uniform enlargement of the organ, due to its becoming infiltrated with fatty elements. As a rule, there is no actual degeneration of the true hepatic structure. It is an intercurrent affection, being associated with gastro-intestinal catarrh, tuberculosis, or other wasting disease. The excessive use of saccharine or starchy food, particularly where associated with a sedentary life during childhood, is also an etiologic factor.

**Symptoms.**—There is a uniform enlargement of the liver, its surface is smooth, and palpation causes no pain. The surfaces of the organ are smooth and soft, and its edges rounded. The general symptoms are negative.

The **treatment** should consist in strict attention to the hygiene and diet. Foods containing large quantities of carbohydrates must be excluded. The patient should take plenty of outdoor exercise, or, where this is impossible, should receive massage. A large part of the treatment consists really in treating the disease from which the fatty infiltration arises.

### AMYLOID DISEASE OF THE LIVER

**Synonyms.**—LARDACEOUS LIVER; WAXY LIVER; SCROFULOUS LIVER; ALBUMINOUS LIVER

Amyloid disease consists in a degeneration of the liver structure, caused by deposits of an albuminoid material the microscopic appear-

ance of which resembles starch granules. The disease may appear at any age of childhood, and in the majority of cases occurs in the course of suppurative diseases, particularly those of a chronic nature, such as tuberculosis, especially tuberculous bone affections, syphilis, and rachitis. It is frequently associated with amyloid disease in other organs. German authorities with large pathologic experience, however, assert that the liver is rarely affected in childhood.

**Pathology.**—The liver presents a pale, glistening, anemic appearance and has a doughy consistency. There is a uniform enlargement of the organ. There are certain chemic tests whereby the presence of amyloid degeneration can be easily ascertained. One of these is made by first cleansing the surface of the organ and then brushing over it an aqueous solution of iodine with iodide of potassium. Wherever the deposits of amyloid substance have taken place these will assume a brownish or mahogany color, which in turn will change to violet or a bluish tint if diluted sulphuric acid is added. Another test consists in brushing the liver over with a solution of anilin violet of the strength of 1 per cent. This will produce a red or pinkish color on coming in contact with the amyloid deposits, the unaltered tissues being stained blue.

**Symptoms.**—Anemia is usually the most prominent symptom. There may be some prominence of the external abdominal veins. Where jaundice is present it is slight, but in the majority of cases it does not appear. Diarrhea is generally a prominent symptom, and hemorrhage from the bowels may occur. Dyspepsia is common. Where the kidneys share in the amyloid degeneration, dropsy in various parts of the body may occur. In these cases the urine will be increased in amount and will contain albumin. Palpation will show that the liver is greatly increased in size, sometimes attaining two or three times its normal dimensions. This enlargement is uniform. The edges of the organ will be found to be round and hard, and palpation, as a rule, does not give pain. Enlargement of the spleen is generally a coincident symptom.

**Diagnosis.**—Amyloid disease may be suspected in any case where increase in the size of the abdomen occurs in the course of a chronic suppurative disease. In suspected cases examination of the kidneys and spleen should be made, and where these are found to share in the amyloid degeneration, diagnosis is positive. Intended surgical operations on the bones and joints should not be attempted during the course of this disease.

The **prognosis** is unfavorable; although the affection may proceed either rapidly or slowly, the termination is always fatal.

**Treatment.**—Outside of the fact that all effort possible should be made to bring the patient's system into as good a condition as possible by the use of tonics and the best hygienic surroundings, treatment should be largely symptomatic. Various drugs have been recommended, particularly chlorid of ammonia and syrup of the iodid of iron. Iodin has also its advocates, but in the majority of cases dependence upon any of these agents will be found to be disappointing. Efforts should be made to remove the cause as quickly as possible, especially where this arises from any suppurating disease of the bones. In cases where congestion of the hepatic or portal circulations due to cardiac weakness occurs, such drugs as digitalis, strophanthus, or other heart tonics will be found of service.

### ACUTE YELLOW ATROPHY OF THE LIVER

Yellow atrophy is very rare in childhood. At this period of life the etiologic factors are usually syphilis or poisoning from phosphorus. The disease is also known by the names of general parenchymatous hepatitis and malignant jaundice.

**Pathology.**—At first there is hyperemia of the hepatic cells, with a grayish exudation between the lobules; following this the cells undergo fatty degeneration. A marked reduction in the size and weight of the organ occurs. The spleen is enlarged, and degeneration of the kidneys takes place. The urine contains bile pigments and albumin, and generally crystals of leucin and tyrosin will also be found in it. The disease is of insidious onset; there are symptoms of general malaise, accompanied by icterus. In the beginning there is a slight elevation of temperature, some pain over the epigastrium, headache, nausea, vomiting, and a coated tongue. As the disease progresses the headache becomes worse—indeed, is generally a prominent symptom—the pulse becomes slow, and the jaundice intense. Later, the patient shows all the symptoms of profound toxemia. There may be fever, or occasionally a subnormal temperature. The stools are black and tarry, and vomiting of black, grumous substance, the so-called coffee-grounds vomit, may occur. In the last stages the patient becomes comatose or may die in convulsions.

The **treatment** is chiefly symptomatic; efforts should be made to relieve the toxemia by hot packs, hot drinks, and the administration of calomel or other agents that will act on the liver. Diuresis should be encouraged. Small doses of phosphorus have been recommended, but have so far proved of no avail.

## HYDATID DISEASE

**Etiology and Symptoms.**—Although hydatid disease is a rare affection, in the temperate zones, it is occasionally seen in children. In Iceland, and in the tropics, where children are closely associated with dogs, hydatid disease is of fairly frequent occurrence. The methods of infection are the same as in the adult. The first symptom noticed is generally enlargement of the abdomen, the tumor being greatest in size in the region of the liver. This enlargement of the hepatic region may be general, although usually the greatest amount of swelling corresponds to the position of the cyst. If this is situated on the convex side of the liver, percussion will demonstrate that the normal area of dullness extends higher in the axillary region in front and in the scapular regions behind. If the cyst is situated in the right lobe, dullness will be found to extend downward toward the umbilicus, and when in the left lobe, examination will reveal the presence of the greatest amount of swelling in the epigastric region. The tumor is usually painless, and percussion over it will cause little or no discomfort. Fluctuation may be found, and occasionally the symptom known as hydatid fremitus may be detected by placing one hand over the tumor and forcibly and quickly tapping another part of the tumor with the other hand. There is generally little or no deterioration in the general health so long as the cyst remains unruptured or there is no great pressure upon the hepatic duct. Jaundice is either slight or, where it does appear, it develops slowly. Rupture of the Griffith cyst is generally followed by symptoms of pyemia, and whenever in the course of development of a hydatid cyst periodic elevations of temperature arise, these being preceded by rigors and followed by sweats and general prostration, rupture of the cyst can generally be diagnosticated. This rupture may take place into the hepatic duct or, in some cases, into the surrounding organs, such as the pleura, colon, bronchi, or even into the pericardium or vena cava.

The **diagnosis** is principally made from the character of the enlargement of the liver. An irregular increase in size, the presence of fluctuation, the absence of pain on palpation, and the continuance of a fairly good condition of general health serve as diagnostic points. From hepatic syphilis hydatid disease must be distinguished by the fact that in the former the enlargement, although present, is irregular, the liver is harder, and some of the general symptoms of specific disease will be present. The diagnosis between hydatid cysts and abscess of the liver is often one of extreme difficulty. In abscess there is usually

a history of traumatism; there are pain and tenderness over the liver, and the general symptoms are more rapid in their onset. Hydro-nephrosis may in some cases be mistaken for hydatid disease, but the former is very rare in children. When doubt exists, an exploratory puncture will aid in diagnosis. Affections of the pleura may be in some cases confounded with hydatid disease, and the diagnosis may be rendered particularly difficult where a cyst has ruptured into the right pleural cavity. In the latter case probably the only point of differentiation will be the presence of the hooklets of the echinococcus in the sputum.

The prognosis is variable. A fair number of cases will probably go for years with one or more of these cysts gradually developing in the liver. Indeed, cases have been reported in which spontaneous cure has occurred. When the cyst is in such position that it can be attacked by operative measures, its removal is recommended by most authorities.

**Treatment.**—It is generally considered that the best results in treatment are obtained by surgical means. The abdomen should be opened at the most prominent part of the tumor, and the cyst evacuated. If any small cysts exist, they should be carefully emptied at the same time and a thorough irrigation of the abdominal cavity should be practised. It is generally recommended that the drainage-tube be left in the incision. Great care must be taken to remove every part of the cyst, as suppuration will take place if any portion of the original cyst or daughter cysts is allowed to remain. Where the cyst is situated in the upper surface of the liver, it is sometimes necessary to open it through the diaphragm; in this case one or two ribs must be resected and the pleura stitched to the diaphragm.

Electricity and injections into the cyst have been tried, but have not met with much success. In cases where, for any reason, laparotomy cannot be done, it has been advised by some that the cyst be opened by means of a large trocar and cannula, and a drainage-tube inserted through the latter. Simon's method consists in passing needles into the tumor, so as to cause an adhesive inflammation, and then later to practise incision. Aspiration has also been followed by temporary success and has been recommended by Dieulafoy and Murchison. Even after operative treatment recurrence may take place, even as late as two or three years after incision, as in two cases reported by Morris. Biliary fistula is also one of the secondary dangers of operative treatment, and cases of this have been recorded.

## CHAPTER VIII

### ABDOMINAL TUMORS OF INFANCY AND CHILDHOOD

The abdominal neoplasms of early life are of rare occurrence. How rare may fairly be gathered from the experience of one of us, an experience covering twenty years of active hospital and private practice among children. The cases may be briefly summarized as follows:

#### SARCOMA OF THE KIDNEY, FOUR CASES

One patient, a five year old girl, came to the Polyclinic Hospital in September 1898. She had a large abdominal tumor and was markedly cachectic. Her case was diagnosticated one of sarcoma of the left kidney. She was operated upon by Thomas S. K. Morton, who removed the tumor mass *in toto*. She suffered little shock, made a good recovery, and one month later left the hospital in blooming health. Recurrence occurred with frightful rapidity, the site of the healed wound eventually opening. Metastatic growths also appeared in both pleuræ. She died early in December of the same year. Upon histologic examination, the tumor proved to be an endothelioma.

The second patient, a girl three years old, presented an enormous growth apparently occupying the greater portion of the abdominal cavity. Nevertheless, the tumor mass could be definitely distinguished from the liver by the determined relations of the ascending and transverse colon. The superior pole of the mass was rotated forward so markedly, that involvement of the retroperitoneal lymph nodes was surmised. The preoperative diagnosis was sarcoma of the right kidney and of the retroperitoneal glands. An operation performed by Dr. John Gibbon verified this diagnosis. After the right kidney had been completely extirpated, handfuls of soft sarcomatous lymph nodes were removed. Very naturally, the patient succumbed to postoperative shock. The tumors proved to be of the small round-cell variety.

The two other patients were lost sight of, after their parents had refused operative interference. Both diagnoses seemed clear, however, and both were verified by other workers in pediatrics. It is too often the case that these patients are taken from one clinic to another in the vain search for a favorable opinion; and so much valuable time is wasted that all hope for a favorable operative outcome is lost.

## SARCOMA OF THE LIVER AND OF THE RIGHT SUPRARENAL BODY.—

The hepatic growth in this case was enormous, filling the abdominal cavity. When one of us saw the patient, she was but seven weeks old. But one opportunity for study was presented, and the tentative diagnosis made was sarcoma of the right kidney. It was an inexcusable error, for the lower border of the liver was clearly felt and remarked upon in the clinical notes. The case has been fully reported by William Pepper, Jr., who made the autopsy and found a large sarcoma of the liver. When Simon Flexner was told of this autopsy, he said: "Primary sarcoma of the liver does not occur." When he was shown the specimen, however, he frankly remarked: "That is a sarcoma of the liver, and it looks like a primary one." Further study, however, lent proof to the correctness of his initial position; for the primary growth was detected in the right suprarenal gland. This experience led Pepper to make his classic study of the relationship between suprarenal sarcoma and congenital sarcoma of the liver.

DERMOID CYST OF THE OVARY.—This growth occurred in a seven year old girl. This child was studied by a large number of medical men and surgeons. All but one concurred in the opinion that she had a sarcoma of the left kidney. One man, noting the cystic fluctuation, and being influenced by the high position of the mass in the abdomen, viewed the tumor as a cyst of the spleen. The passage of the transverse colon in front of the mass, made the rest of us feel sure that it was not a splenic tumor; though subsequently, it did prove to be a bilocular cyst. If the writings of Howard Kelly and George Carpenter upon rectal examinations in childhood had then been extant, the mistake in diagnosis would scarcely have been made. Indeed, the late Frederick Packard sounded a note of caution when he made a rectal examination and remarked that he could feel no mass posterior to the examining finger. However, he concurred in the diagnosis of renal neoplasm. Indeed, the position occupied by the mass, high in the abdominal cavity, made us fail to consider the possibility of an ovarian growth. On operation, a large dermoid cyst of the left ovary was removed by T. S. K. Morton. The specimen presented two cysts, joined to one another by a relatively narrow isthmus of solid tissue, thus producing a wonderful simulation of a kidney's shape. The specimen contained skin, hair and an imperfect mandible.

TUBERCULOUS PERITONITIS WITH A ROLLED UP AND THICKENED OMENTUM.—Two cases of this type have been seen. Strictly speaking, of course, these were not cases of abdominal tumor; but in both, after careful studies, diagnoses of renal sarcoma had been made.

As the same mistake has been made by others, it is thought well to cite these cases in this section. Both children were subjected to rectal examination, and in the study of one of them, a skilled röntgenologist secured a reniform shadow that made him willing to stake his reputation upon the diagnosis of renal growth.

It has also been our privilege to see the beautiful specimen of sarcoma of the small intestine removed from a child by Wm. E. Robertson. Again, we saw the operation for hypernephroma performed by W. Wayne Babcock upon a patient of Henry Jump's. Jump had made a splendid study of this case, a girl exhibiting a masculine voice, precocious puberty and false hermaphroditism (the clitoris resembled a male organ). He had correctly diagnosed her sex, and had suspected the adrenal character of the neoplasm.

**Etiology.**—Abdominal tumors may appear at any age of infancy or childhood. Indeed, congenital cystic kidneys and renal sarcomata have interfered with or prevented birth. Most cases are observed in infancy or in early childhood. In our own small experience, the female sex has furnished most of the subjects.

We know no more and no less concerning the causes of abdominal tumors than we know of morbid growth production in other regions of the body. On the other hand, the factors known to bear a relationship to tumor growth in general are all found to be operative here: Thus, inclusions are recognized as responsible for dermoid cysts or actual teratomata (fetus within fetus) of the abdominal parietes, ovary, etc. Retention cysts may originate in the stomach, intestine, kidney, bladder, ovary, etc. Injury may bear a relationship to the rare pancreatic cysts, the very rare carcinomata of early life, or to the acceleration of sarcoma growth in various abdominal tissues. Parasites are responsible for the occurrence of the echinococcus cysts of the liver, peritoneum or bowel; abnormal growth of embryonal tissue is discernible in the development of the sarcoma and the hypernephroma.

**Pathology.**—Sarcoma is far and away the most common abdominal tumor of early life, and the kidney is far more often affected than any other abdominal organ. This statement is true of temperate zones. In Iceland and in the West Indies, where children are thrown in close contact with dogs, the literature would appear to show that echinococcus cysts are the most common growths. We have seen but one case, and the patient was not a child. He was a youth 19 years old who was studied by the late Dr. Musser at the Philadelphia General Hospital. Far more rarely, however, benign or malignant tumors may develop in the liver, pancreas, spleen, stomach,

intestine, peritoneum, omentum, adrenals, ovary, uterus and bladder. For a full discussion of the various neoplasms that may involve the abdominal parieties or intra-abdominal organs, the reader is referred to works dealing with special pathology.

**Symptomatology, Physical Signs and Diagnosis.**—To give a full clinical description of the different abdominal tumors of childhood, and to make complete diagnostic differentiations between them, would necessitate the writing of an exhaustive monograph. It is with the hope that the following tabulations will prove suggestive to the student that they are submitted.

Rectal examination, particularly rectal examination under anesthesia enables the examiner to outline the internal genitalia of the little girl. He may also detect prostatic growths in boys. In suspected neoplasms of the bladder (with vesical irritation, hematuria, etc.), skilled cystoscopic examinations may prove invaluable. They are difficult to make in boys, however.

**Prognosis.**—This depends upon the size and character of the growth and the nature of the treatment followed out. At best, it is a sorry chapter. Benign growths, as in a patient of Israel's, may persist for years, even though they attain enormous size. The only hope in the presence of a malignant neoplasm is early and complete surgical removal. Even then, as Robert Abbe found, "three years of quiescence may be followed by a return of the growth."

**Treatment.**—This is surgical. As soon as a diagnosis of abdominal tumor is made, operation should be performed for purposes of exploration and with the hope that the growth may permit of complete extirpation. After such an operation, the child should be submitted to X-ray treatment for a considerable period of time. There is much difference of opinion concerning Coley's method of dealing with inoperable malignant growths; but the few signal successes secured would seem to render such treatment justifiable in the presence of inoperable growths.

(For fuller information upon these subjects, the reader is referred to the writings of Israel, Bland Sutton, Robert Abbe, Kelynack, Watson, etc. A full bibliography may be found in the "Index Catalogue of the Surgeon General's Library.")

Nature of the neoplasm	Symptomatology	Inspection	Palpation	Percussion and auscultatory percussion	Röntgenology	Urine	Stools	Blood	Exploratory puncture
<b>Kidney, adrenals and retroperitoneal-lymph-nodes.</b>	May not be in evidence. Cachexia in rare cases of carcinoma. Cachexia usually late in kidney, hydronephrosis, etc. Precocious puberty and masculinity (in girls) in the presence of adrenal growths, or heterotopous adrenal tissue (hypernephroma). Pain may be marked, slight, or even absent.	Bulging in the corresponding hypochondriac lumbar and iliac regions. Posterior lumbar bulging is very characteristic. Enlargement of the superficial abdominal veins. No movement of the mass in respiration. Peristaltic movements may be noted in the ascending or descending colon, surrounding the crest of the growth anteriorly. The mass may fill the whole abdomen.	Bimanual palpation, with one hand posteriorly placed, reveals a mass of great weight and density (unless cystic). The mass is reniform in shape. Rectal palpation, with one finger, may prove that the growth is posterior. Fluctuation may be detected in cysts.	Confirms the density as determined by palpation. Right-sided renal growths displace the ascending colon toward the median line, while left-sided ones similarly displace the descending colon. (This never occurs in hepatic or splenic enlargements.) Fluctuation may be detected by percussion. By auscultatory percussion, the dullness of the mass may be differentiated from the splenic or hepatic dullness.	A reniform shadow may be detected that can be differentiated from the liver shadow. This should not be depended upon as a sole method of diagnosis.	Said to contain blood ("shadow-corpuscles") at some time in half the cases of renal sarcoma. Blood may appear as late. The examination of a 24-hr. specimen is most valuable.	Normal, unless the growth is large enough to produce mechanical obstruction. In intestinal growths, on the contrary, the obstruction occurs early and the stools may contain blood.	Leukocytosis of a slight grade may appear in the presence of a small round cell sarcoma. The lymphocytes are relatively increased in echinococcus cysts.	Not justifiable unless the tumor is cystic. It should then be performed posteriorly. Urine may be obtained, or "hooklets" in echinococcus cysts.

Hepatic growths.	Nature of the neoplasm	Symptomatology	Inspection	Palpation	Percussion and auscultatory percussion	Roentgenology	Urine	Stools	Blood	Exploratory puncture
	<p>Like the spleen the liver may be enlarged in many constitutional conditions (rickets, syphilis, etc.). Anomalies of the bile ducts may also cause enlargement. These conditions must be carefully distinguished from true neoplasms. Non-malignant growths are exceedingly rare; but the echinococcus cyst is seen in some countries. Sarcomas are rare and are probably always secondary. Carcinoma is of extreme rarity.</p>	<p>Jaundice may occur late in cystic growths. Cachexia is rare, even in constitutional carcinoma, because of the extreme rapidity of growth. Pain may be present.</p>	<p>A mass, conforming to the shape of the liver, in the epigastric, right hypochondriac, and right iliac regions. This does not protrude posteriorly as renal tumors do. The mass may be irregular with cystic tumors or gummata. The lower border moves downward in inspiration.</p>	<p>Confirms inspection in respect to shape, extent and movement of the mass. Light palpation conveys more information than any other single method. Fluctuation obvious in cysts. Rectal palpation yields only negative results, but may serve to differentiate from post-peritoneal growths.</p>	<p>Light percussion most valuable. It gives more information, particularly concerning resistance. It confirms inspection and palpation concerning the shape and size of the mass, and palpation concerning its density or fluctuation. The ascending and transverse colon are never detected in advance of the growth, just as the transverse and descending colon are never in front of splenic tumors.</p>	<p>The shadow may correspond to the shape of the liver.</p>	<p>Bile salts and pigments if jaundice is present.</p>	<p>Acholic, if the bile passages are obstructed.</p>	<p>?</p>	<p>Hooklets in echinococcus cysts. Pus in amebic or subdiaphragmatic abscess. Blood only in malignant growths.</p>

	Nature of the neoplasm	Symptomatology	Inspection	Palpation	Percussion and auscultatory percussion	Roentgenology	Urine	Stools	Blood	Exploratory puncture
Peritoneum.	Non-malignant cysts. Primary sarcoma and carcinoma (very rare). Secondary malignant growths may invade from the contiguity of affected organs.	With cysts there may be no symptoms. Cachexia with malignant growths. Fever, rapid pulse and probable involvement of other organs with tuberculous peritonitis. Emaciation and hectic in tuberculous peritonitis. Pain may be present.	May simulate in form and resistance other growths. Usually diffuse swelling. Cutaneous and percutaneous tuberculin reactions may be applied and observed. Prominent abdominal veins. Possible "pouting" of the umbilicus.	Board-like mass felt rather superficially. Shape may simulate other large mesenteric or inguinal glands may be palpated. Rectal examination may detect a mass anteriorly.	Board-like resistance. Intestinal tympany absent, at least over the mass. Usually uniform dullness.	Usually negative evidence only. Shadows may mislead	Not characteristic unless nephritis or amyloid change accompany tuberculous peritonitis.	May be loose and foul in tuberculous peritonitis. Intestinal obstruction may result from neoplasms.	Lymphocytosis in tuberculous peritonitis.	This should not be performed unless we feel sure of fluid in a cyst or free in the peritoneal cavity.

## CHAPTER IX

### DISORDERS OF METABOLISM

#### RACHITIS

Rachitis, or rickets, is a disorder of nutrition (innutrition or malnutrition) dependent upon dietetic and hygienic causes. While its characteristic and essential pathology is confined to the bones, it is capable of affecting every tissue in the body. If the modern pediatrician were asked upon what tissue it exerted its most malign influence he would probably reply "upon the nervous system."

Rachitis is a far more common condition than statistics would lead us to believe. It is at the foundation of much of the impaired resistance in infancy and early childhood. It seems one of the ironies of fate that so common and important a disease should be so little known to parents.

**Etiology.**—Many theories have been advanced to explain the occurrence of rickets, but none has done so with perfect satisfaction. We may safely say in a general way, however, that it is primarily dependent upon dietetic and secondarily upon hygienic causes. The dietetic factors of importance are insufficient quantities of fat and protein in the food. Thus it often develops in artificially fed babies, when carbohydrates are used in excess to replace the deficits in the other organic foods. Housing, with its insufficient fresh air and sunlight, seems to be of importance in many cases.

Rachitis is found in many countries, chiefly in cities or in crowded, underfed communities lacking light and air, and is rare in country places or where the food-supply and hygienic conditions are favorable. It is more common in Europe than in America, and our supply of cases of the disease is augmented annually from there. It is shown by Morse and others to be on the increase in this country. Observers in the large clinics of Europe admit its appearance in over 30 per cent.<sup>1</sup> of children applying for advice. In this country it is not quite so common or severe, even in our largest cities, and is comparatively rare out of crowded centers unless brought thence. With us in America it is seen either in the children of emigrants (Italians and Armenians),

<sup>1</sup> Some foreign authorities claim that 50% of all infants show some evidences of rickets, and that 90% of city babies are affected.

or in the negro and mixed races, often in babies who have been hand-fed or who early get coarse or unsuitable food, particularly when this is deficient in fat. Snow has called attention to the fact that here it is most noticeable in the transplanting of a southern race to a northern climate. It appears now and again in the families of the well-to-do, even among the rich, especially where nervous mothers coddle their children overmuch or practice erroneous methods of feeding and nursery hygiene, above all if enough air and sunlight are denied them. Rickets is rarely congenital, most of such cases being instances of achondroplasia foetalis, and late rickets is also an exceptional occurrence. Its gross bony manifestations are usually manifested between six months and two years of age; but the disease may often be recognized much earlier, giving opportunity for therapy.

Predisposition probably has much to do with the production of rickets. The offspring of parents in feeble health, overworked and underfed, with poor digestion and assimilation, a senile father or a mother from any cause exhausted, as by prolonged lactation, discharges of pus or blood, etc., is apt to be rachitic. Rachitis is not distinctly hereditary, although at times it has the appearance of being so.

Parental syphilis, alcoholism, and tuberculosis probably induce rachitis. Infants who begin life with an unfavorable inheritance, and who are confined in dark, filthy, overcrowded houses, especially in damp cellars, are very prone to develop the disease; even those who begin with good constitutions may thus become rachitic. The most powerful factor is food deficient in certain essential qualities, as fat and proteins. Breast-fed infants usually escape, but not always, and those dependent upon breast milk from poorly nourished mothers or upon the parental supply for too long a time are liable to the disease. Even where the supply of food is good and yet the digestion is too weak to cope with it, infants may thus be affected. Those infants who have been fed on the proprietary foods are especially prone to this disorder. If the strength be reduced by any cause, such as summer diarrheas, rachitis may begin without any intervening stage (Eustace Smith). Rickets is a frequent sequel of exhausting infantile disorders, especially in one predisposed.

**Pathology.**—While true that the essential and most conspicuous changes are observed in the ends of the long bones, the morbid anatomy is that of a constitutional disease, a blood dyscrasia, affecting the nutrition of nearly all tissues of the body. The primary lesion is hyperemia of the periosteum, the marrow, the cartilage, and of the bone itself. The changes in the bones may be classed as microscopic and

gross changes, but neither group can be thoroughly understood without an understanding of the normal processes of ossification. The long bones grow in length from their epiphyses, or better, epiphyseal cartilages (endochondral ossification); they increase in thickness from the subperiosteal changes (subperiosteal ossification). Synchronous with this new bone formation without, reabsorption of bone already formed is also proceeding from within, leading to the formation of medullary spaces in the red marrow and of the central canal in the shaft of the bone. Practically, a microscopic examination detects the following layers as ossification proceeds at the epiphysis: A. Normal hyaline cartilage. B. A layer of cartilage proliferation. C. A layer of columnar formation of the proliferated cartilage cells. D. A layer of calcification. E. A layer of ossification, with the formation of the normal Haversian systems. Blood-vessels protrude into this newly-formed tissue from the diaphysis, and osteoblasts are found in abundance. Similar changes are taking place under the periosteum of the diaphysis. Osteoclasts are active in the reabsorption of bones. In rachitis of any severity one observes the following departures from these normal processes. *Layer B*: There is undue proliferation of the cartilage cells and they may be unusually large. They tend to stain poorly. *Layer C*: The formation into columns is most imperfect, though the columns may be more numerous. *Layer D*: Calcification is most imperfect, being often observed but in islands. The calcium salts may be reduced in amount from 30 to 50 per cent. Holt states that the ratio between the organic and inorganic materials (1 to 2) may be reversed. *Layer E*: Ossification is still less perfect than calcification. Nor are these different layers clearly separable as in normal bone; for one finds all of these imperfect stages in the same layer. Undue vascularity also obtains. While the osteoblasts are failing of their bone constructive function, the osteoclasts are more than normally active in their reabsorption rôle. It seems as though nature had expended her best efforts in the early stages of the process (cell proliferation) and had paused exhausted in the later ones; that then, dissatisfied with her product, she had proceeded to destroy it.

These departures from the normal once understood, the gross changes become readily explicable. The epiphyses are enlarged and the diaphyses thickened because of undue cell proliferation and undue vascularity. Cranial bosses or prominences are produced in the same manner, while craniotabes represent an unduly active process of destruction (reabsorption). The lessened amount of mineral matter (imperfect calcification) and the still less perfect bone formation

render the bones unduly soft; hence, deformities are readily induced by the following forces: The growth of underlying soft tissues (brain, etc.), atmospheric pressure (thorax, etc.); muscular force (Harrison's groove, deformed clavicles, etc.); weight of the extremities; superimposed weight of the body (deformities of spine, pelvis, extremities, etc.) In the repair of these diseased tissues nature often overdoes her work and the thickened and deformed bones become unduly hard (eburnation).

The prominent visceral change is the enlargement of the spleen and liver. The splenic enlargement is not a primary but a secondary process, possibly due to toxic disturbances, the result of some essential process. At first the enlargement is due to an increase in the pulp, later in the connective tissue. Starck found the spleen enlarged in over one-half of the autopsies on rachitic children and in 68 per cent. of living cases.

The liver is less frequently enlarged, though it may more often appear so through displacement and distortion resulting from the thoracic deformity. The visceral pleuræ and lungs often exhibit deep impresses from the costal beading and the "Harrison's groove." Emphysema may appear in adjoining areas. Bronchitis is commonly observed, and as complications one may find areas of catarrhal pneumonia, atelectasis or tuberculous disease. The hollow viscera are usually much dilated and visceroptosis may be observed. Catarrhal states of the gastric and intestinal mucosa are also the rule. Adenoids are often present and hypoplasia of the various lymph nodes is also the rule. Changes have been observed in the kidneys, though they are not characteristic nor essential. Hydrocephalus and hyperplasia of the brain have been described by certain authorities. The blood changes will be described under the symptomatology.

**General Symptoms of Rachitis.**—The first symptoms of rickets in a child are fretfulness, disturbed, intermittent sleep, and slight fever at night. The little patient becomes mildly cross, repelling advances, its pillow becomes wet with sweat during sleep, beads of perspiration appearing first on the forehead and face. It seems to suffer from a sensation of heat or oppression, and kicks and throws off the bedclothes, inducing a peril from chilling of the areas thus exposed, especially as the surface is usually abnormally damp and the skin relaxed. There is often marked pallor, and a diffuse soreness and tenderness of the body. The indisposition on the part of the little sufferer to be moved, however, is not due to the tenderness of the joints, unless scurvy be present, but rather on account of the respiratory distress

induced. The digestion is often disturbed, flatulence, fetid stools, constipation or rarely diarrhea, with evidences of intestinal catarrh being common. The appetite is often ravenous, especially for meat and fatty substances. The digestion is slow, however, and imperfect, and food is passed undigested, the stools having a most abominable odor, often curdy-looking and surrounded with mucus. Aaron Jacoby contends that constipation is one of the earliest symptoms of rickets (dependent upon atony of unstriated muscular tissue), and this accords with our experience. Dentition is often delayed until the ninth or tenth month or even beyond the first year of life.

There is probably no disease of early life that affords the student such splendid opportunities in physical examinations as the disease rickets. It is our custom to use it as a model in illustrating the method to be pursued in the objective examination of the sick infant. Not every tissue is affected in every case, nor is the degree of the affection always the same; nevertheless, the rachitic process may exhibit characteristic phenomena from the *crown of the patient's head to the soles of his feet*. Let us again pursue such an examination. The hair is often lusterless and dry, and the occipital bald spot is commonly observed. If the child is observed during sleep, however, sweating of the forehead and scalp may immediately attract our attention. The skull is usually square (brachycephalic) and frequently asymmetric. If the posterior parietooccipital region is unduly flattened on one side, the frontal bone is prominent upon the same side. The fontanelle is unduly open and its closure is delayed until two years of age or beyond that period. Cranio-tabes (undue softness of the bone, giving to the examining finger an elastic cartilaginous sensation) may be detected in the region of the anterior fontanelle or along the lambdoidal suture. It is more commonly detected during the first six months of life. On the other hand, marked prominence of the frontal and parietal eminence may give rise to the type of skull that is known by the following names: Natiform type, four-hill variety; "hot-cross-bun type," etc. In contradistinction to the square type of skull, the doliocephalic rachitic cranium may be seen. The eyes exhibit nothing characteristic; but because of nasal obstructions and catarrhs, inflammatory conditions of the conjunctivæ or cornea may result. The nasal bridge is often broadened and the anterior nares constricted, while coryza is common. Adenoids are more common than in non-rachitic babies. Dentition is delayed as mentioned and the milk teeth when they appear are often deficient in enamel. Irregular dentition is almost pathognomonic. Even the permanent teeth may display distortions and serrations that tell of past

rickets. The maxilla is unduly lengthened (sagittal diameter) and its anterior angle is acute. The mandible is also deformed in a characteristic manner, being shortened and squared (flattened anteriorly). No doubt the adenoids and the frequent habit of thumb sucking contribute to these jaw-bone deformities. The faucial tonsils are often hyperplastic. Middle-ear disease readily occurs because of the adenoids. As previously mentioned, the lymph nodes, particularly the various cervical chains, are often palpably enlarged. The clavicles are markedly bowed and sometimes their sternal ends are actually higher than the acromial. We have seen the chin almost resting in the suprasternal notch.

The thorax is usually flattened laterally and the sternum unduly prominent (chicken breast). Depression of the xiphoid region is not uncommon and this may amount to the "funnel-shaped" deformity ("trichter-brust"). Laterally, from the ensiform region, extends a deep furrow, the well-known Harrison's groove. It is produced by the action of the diaphragm on the unduly soft ribs and cartilages. The ribs usually flare out below this groove. Probably the term "violin-shaped chest" (Tyson) best describes the complete appearance. Beading of the ribs is probably one of the most common and also one of the earliest objective finds. It produces the well-known appearance of the "rickety rosary." Internal beading is more marked than its external manifestation. (See pathology.) Dyspnea and hypernea are commonly observed. Bronchitis is common and pneumonia and tuberculous disease, as mentioned, too often attack the physically cramped and functionally handicapped lungs. Even the aorta has been deformed (Jacobi) as a result of chest malformations.

The abdomen is usually protuberant ("pot-belly"), though often the prominence disappears in the supine position. This prominence is probably dependent upon two causes, viz. hypotonia of the abdominal muscles and distention of the hollow viscera. There is little doubt that that visceroptosis plays a part in some cases. Umbilical hernia is often present, and when not congenital, is a result of the muscular hypotonia. The same statement may be made concerning diastasis of the recti muscles, a condition that is most frequently observed superior to the umbilicus.

When muscular effort is made (as in change of posture) a protrusion is noted in the median line between the separated recti. If the fingers of the examiner are placed in the sulcus between the edges of the recti, similar efforts will result in a grasping of the same. ("Webster's sign.")

Inguinal hernia, too, is not uncommon, though far less frequent than the umbilical variety. Palpation and percussion will usually enable one to detect splenic enlargement, and frequently enlargement or misplacement of the liver. Percussion and auscultatory percussion may elicit evidences of gastric and colic distention. Turning to the back one usually finds a lower dorsal or dorso-lumbar kyphosis (rachitic curve) which is "rounded out" rather than acutely angular as in Pott's Disease. Unless permanent changes have taken place, this curve is only seen in the sitting posture. When the subject is erect, a lumbar lordosis is apt

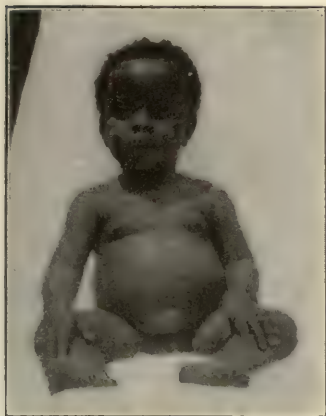


FIG. 35.—A MARKED CASE OF RICKETS IN A 2-YEAR-OLD BABY. He has a doliocephalic skull with prominent bosses. The "occipital bald-spot" is well marked. He has adenoids, a general adenopathy, a deformed chest with a marked Harrison's groove, a pot belly, an umbilical hernia, an enlarged liver and spleen, enlarged epiphyses of the long bones and rachitic paraplegia. He has but 4 teeth.



FIG. 36.—A RACHITIC DWARF 12 YEARS OLD. Syphilis has probably played a part in his case, as he has "sabre shaped" tibiae.

to be observed. Scoliosis also occurs, though it is more rare. The pelvis is frequently deformed, and the importance of such deformities in female children, can scarcely be overestimated. The most common one is the flat rachitic pelvis. Obstetrically, it may acquire dire significance in later life. The extremities in advanced cases, may reveal many deformities, though these are usually not observed until the second year of life. They presuppose superimposed weight, faulty postures, etc., for their production. The principal ones are knock-knee,

bowed-legs (bow-legs), flat foot, bowed-femora and bowed radii. Green stick fractures may occur in the severe cases. Epiphyseal enlargements, usually most marked in the radii and ulnari are also observed. The rachitic hand presents the so-called "bead-like" fingers, which are rounded out and more prominent between the interphalangeal joints than at the joints themselves.

Pallor of the skin surface and mucous membranes is usually present, and the skin is unduly leaky. Upon slight exposure to cold,



FIG. 37.—MARKED BOWING OF THE LEGS IN A RACHITIC SUBJECT.

peripheral cyanosis is often noted. In uncomplicated rachitis, the subcutaneous fat may be very abundant, even unusually so. Despite this fact, however, the flesh feels unduly soft, and in picking them up one is surprised to find they weigh so little. One of our nurses styles these fat rachitic babies "little-puff-balls."

The blood exhibits the characteristics of a secondary anemia, with the hemoglobin usually reduced to a greater degree than the number of corpuscles. Poikilocytes may be observed. In the severe cases, a moderate leukocytosis is usually present.

Muscular hypotonia is the rule, and in bad cases considerable atrophy may obtain. The nervous phenomena are numerous and important. From the motor side, one should first note, retardation of development. This may be true of sitting, crawling, creeping and walking. The last, particularly, may be delayed until the baby is two years old or more. The legs may appear so weak and useless that the

term rachitic paraplegia is applied. The reflexes are usually present in this false paresis, however, and the electric reactions are normal. Abnormal movements are often present, such as head-nodding and head rotation (gyrospasm). Nystagmus (sometimes unilateral) accompanies the latter condition almost invariably. Tetany, too, when observed in infancy is most commonly seen in rachitic subjects, at any rate in the poorly nourished with digestive disturbances. Laryngospasm (laryngismus stridulus) is rarely observed in other than rachitic infants. The baby so affected suddenly ceases to breathe; it becomes quite cyanosed and death appears imminent. The attack terminates with a long crowing inspiration that is very characteristic. Eclampsia in infancy occurs in rachitic babies far more frequently than in any other patients. We shall find in our future study of eclampsia, that a convulsion implies some underlying or predisposing condition and some exciting factor. The predisposing cause is most frequently rickets. Eclampsia in infancy may merge over into what we are obliged to denominate idiopathic epilepsy; so that rickets as a remote cause of epilepsy deserves consideration. Of sensory nervous phenomena we shall have little to say. It was claimed by the older authorities, and is still asserted by some moderns that rachitic babies are unduly sensitive to handling (hyperesthetic). One should always view this phenomenon as possibly scorbutic until it is proved otherwise. Whether rickets can cause hydrocephalus or not is a moot question. Retardation in mental development is well evidenced in the slowness with which speech is sometimes acquired.

**Prognosis.**—The disease is not fatal of itself, but death often readily results in the enfeebled organism from some intercurrent malady. Under proper treatment recovery takes place, with some resulting deformity.

**Treatment.**—As has been said, we may assume that nearly one-third of all the children of our city population, and perhaps a fourth of others, exhibit the evidence of rickets or may readily acquire the disorder. Therefore, a thorough consideration of detailed treatment cannot be out of place. We will review here an outline of such systematized measures as are suitable, and in the chapter on General Considerations on Physical Development the matter will be treated more fully. The first consideration is prevention; here it is necessary to face the question of accepting or rejecting degenerate immigrants—a matter which is probably about to play a very important part in politics.

The next step is to give our attention to pregnant women who are

of these devitalized classes and races. If this can be done, much may be accomplished. Bathing, good air, sunlight, and abundant, well-cooked food will accomplish much. A short residence in the country would do more. The relief of the coming mother from exhausting labor and overwrought emotion is also of great value. The result of such care may be a pretty fair child, and upon such a little one continued liberalizing influences will work to great advantage, and a few generations of such measures regenerate a moderately good population. The rachitic baby should be kept in a room which faces the south, whence come the best breezes in summer and sunlight at all times. It should be kept at a temperature in the neighborhood of 70° F. (21.1° C.), with the windows much open, or on sheltered housetops or piazzas, wearing extra clothing the while. Underwear should be of wool, thin in summer and thicker in winter; bathing should be daily, as much for tonic action as for cleanliness. If the extremities become chilly after this, or at any time, it is easy and valuable to apply external heat. The breast milk of the mother is always the best food and during lactation she should take tonics and extra food rich in fats and albuminoids. The mothers of the poorer classes fill themselves up on bread and tea while nursing a baby; they had better live on milk and meat. It is important that mothers of babies showing rickets should sacrifice something to the needs of lactation. Good breast milk when insufficient is of little use if supplemented by coarse, ill-cooked "table food." Poor breast milk is better than none, because it can be made better unless the mother be distinctly infected, as by tuberculosis. The natural supply can be supplemented by suitable food better than to depend upon hand-feeding alone. For rachitic babies this extra diet is nearly always necessary, certainly after eight or ten months. The extra food should be cow's milk modified by water, milk, sugar, cream, and an alkali, and possibly some "casein breaker" or attenuant, such as barley or oatmeal water. The milk formulas should first have a low percentage of proteid and a moderately large amount of fat, and later, as the digestion becomes stronger, both these milk elements should be increased. This should alternate with strong animal broths and but little or no starch food. Starch feeding produces rickets in those predisposed, probably not *per se*; but because it replaces the other organic constituents. Weaning should not be allowed in the hot months, certainly not in our large cities. In the preparation of milk the first requisite is proper care as soon as it leaves the cow, and this is receiving most gratifying attention at the hands of dairymen.

If the cow's milk be sound and kept so, it need not be subjected to

heat to fit it for use, except during the summer months. As ordinarily supplied it will require Pasteurization or sterilization; the latter, however, cannot be used but for a short time. Simple boiling over a water-bath does fairly well, especially for older children. Prepeptonization is of great use, particularly for one or two meals in a day and along with supplemental diet.

Properly prepared cereal solutions, as of barley or oatmeal water, are not only cheap and convenient diluents, but of real value to separate particles of casein and prevent lumpy curds in the stomach, and they are in themselves nutritious. They are far better when diastase has been added. The amounts and intervals of feeding are of as much importance as quality. Breast-fed infants of vigorous stock may survive some insufficiency, but the feeble, rachitic infant should receive overfeeding rather than underfeeding. The value of overfeeding or forced feeding in children has not been studied enough except where the food was of bad quality.

It often happens that a very young child, especially a rachitic one, will greedily devour and thrive notably on a liberal diet of beef and mutton.

Constitutional treatment must be instituted in the first half year or year to be efficient. Little can be expected after the second year, for by that time we have chiefly the effects of the disease, not the disease itself, to deal with.

For the deformed chest, and its resultant cramped lungs, regulated gymnastics are indicated. Rarefied air deserves more extended trials. To prevent gross deformities of the pelvis and extremities, much may be done in the way of prevention. The supine position, enforced by appropriate apparatus if necessary, must oftentimes be employed. The muscular apparatus is developed in the meantime by massage, hydrotherapy, etc. Faradic electricity is of value, but is terrifying to the little patient. For kyphosis, extension and postural treatment are indorsed, along with prolonged rest in bed. The treatment of the bony deformities, when they are marked or permanent, belongs to the domain of orthopedics.

**Medicinal Treatment.**—Rachitic children are particularly liable to gastric and intestinal disturbances and weaknesses; their stomachs are usually hyperacid and need alkalies; for the relief of this in the infant pancreatic extract with soda is of use; in older children well-diluted muriatic acid and pepsin are better; also there should be supplied other secretions of the intestines which assist assimilation. It may be well to mention here the malt preparations as being useful

if there be no diarrhea, and to be added to starch food or given alone. The great remedy for rickets is cod-liver oil, not only as a fat, but probably also because of its alkaloids and the iodine it contains. We cannot subscribe to the view that cod-liver oil simply acts as a fat or oil. To cod-liver oil may be added with advantage raw eggs, glycerin, or syrup, and spirits or some heavy-bodied wine or cordial. If we could be sure of getting a cod-liver oil made from fresh livers of fresh codfish, and prepared by cold expression, we would have the best thing obtainable. Some years ago this was forthcoming at the hands of an old sea captain named Stone, of Swampscott, Mass. A favorite preparation of the authors is: Place in an 8-ounce bottle one raw fresh egg; glycerin,  $1/2$  of an ounce; maraschino, curacao, sherry, or port wine,  $1/2$  of an ounce; cod-liver oil, 6 ounces; shake thoroughly, and keep on ice in the dark, and give a dessertspoonful before two principal meals. Russell's emulsion of the mixed fats is of great value. One drug which enjoys a large reputation in overcoming with great promptitude and thoroughness the defects of bone growth in rickets is phosphorus. This should be given in small doses— $1/200$  to  $1/100$  of a grain after meals. There are many authorities abroad and in this country who dispute its value; but in severe cases and in those with marked nervous symptoms it should be given a trial. We believe it a valuable drug. Iron, manganese, and arsenic are of value for the anemia. Animal preparations, notably nuclein and lecithin have appeared to yield striking results in some cases, possibly because of the phosphorus they contained. To relieve sweating, atropin, picrotoxin, and the cardiac tonics are useful, as well as aromatic sulphuric acid.

## ACHONDROPLASIA

**Synonym.**—CHONDRODYSTROPHY FETALIS.

**Definition.**—This is a rare and curious type of dwarfism in which the subject displays an unusual shortness of long bones (particularly of the humerus and femur), a large characteristically shaped head, well-marked lordosis and usually some mental subnormality.

**History.**—The condition is said to have been depicted in Egyptian art. Many of the "court jesters" were subjects of this disease. In his "Cloister and Hearth," Charles Reade gives a splendid description of such a dwarf. The condition, as a pathologic entity, was studied by Virchow and others, but Parrot and Marie gave the first good clinical descriptions.

**Etiology.**—The condition is a congenital one, and its name suggests

its origin. At sometime between the third and sixth months of fetal life, the epiphyseal growth of the long bones ceases or becomes markedly retarded. The cause of this interference in the developmental process is unknown, though sometimes there are evidences in the diseased subject of hereditary syphilis. Premature ossification of the tribasilar bony structures also takes place, so that the bridge of the nose is uniformly depressed, and brain growth takes place in an upward direction. The overhanging forehead is produced in this way.

**Pathology.**—This has been indicated. There is interference with epiphyseal cartilaginous growth, and the columnar arrangement of cartilaginous cells is interfered with. Periosteal bone formation proceeds, and thus the bones grow in thickness. Sometimes the periosteum pushes between the diaphysis and epiphysis, interfering still more with growth in length of the long bones.

**Symptoms.**—Dwarfism of a marked degree; a trunk of normal length for the patient's age; a large globular skull, with prominent bosses; a sunken nasal bridge; a prominent lower jaw; short arms and legs, due principally to shortening of the humeri and femora, and marked lumbar lordosis, furnish the striking features of this disease. The subject also presents the characteristic "trident hand" (main en trident). Usually, these patients are somewhat backward mentally; but most of them succeed in eking out livelihoods.

Many of these children are born dead, and when they survive they are usually weak in infancy. They are very fat as infants; but in adult life may become decidedly muscular.

**Diagnosis.**—The disease is included in this section, because it was formerly viewed as fetal rickets. It has also been mistaken for cretinism. With care, neither of these mistakes should occur.

**Prognosis.**—When infancy is survived, the subject may live a moderately long and useful existence.

## SCORBUTUS

**Synonyms.**—INFANTILE SCURVY; CHEADLE'S DISEASE; BARLOW'S DISEASE; PERIOSTEAL CACHEXIA; SCURVY-RICKETS.

**Definition.**—Infantile scurvy is a disease of early life that is unquestionably dependent upon dietetic causes. Its essential characteristic is hemorrhage, the most common situation of which is under the periosteum of the long bones.

**History.**—Under various names unquestionable cases of this

disease were reported during the latter half of the last century (Möller 1859 and 1862 and Ingersley 1871); but for the true recognition of its character, Wm. Cheadle, of London, deserves credit (1881). Barlow (1883) made important studies of its pathology and to him we are indebted for our knowledge of the subperiosteal hemorrhages so characteristic of this disease. Northrup was the first to direct attention to the frequency of infantile scorbutus in the United States. The collective investigation of the American Pediatric Society (1896) did much to increase our knowledge of this disease.

**Etiology.**—The causes are quite well understood. It is a disease that requires time to produce, probably at least three or four months of bad feeding. Hence, it is rare under six months of age. It is most common between six and eighteen months of age. In contradistinction to rickets, it is a disease of the better classes. The food that is most frequently responsible for its production is a stale food, the proprietary infant food, etc. Next in the order of etiologic importance comes sterilized milk. Very few cases appear dependent upon pasteurized milk, but such have been reported. Low proteid formulæ have also seemed responsible for its production. In a very few cases, it has occurred in breast-fed babies; but the mothers' milks have been found lamentably low in organic constituents under such conditions. Cheadle believes that the citrates and other antiscorbutic elements are absent or changed in stale and sterilized foods. For lack of a better and more correct term we may say that the elements of freshness are lacking.

Is this disease the same disease that is seen in the adult, simply modified by the age of the patient? That is the belief of Cheadle and of the majority of authorities. Is it a separate and distinct disease? That is the belief of some. Is it scurvy-rickets? That is the view of certain English and German authorities. The disease is often seen in rachitic subjects; but it is again observed in patients not presenting the slightest evidence of rickets. Is it an infectious process? That is a view advanced by R. Koch, but not accepted by other authorities.

**Pathology.**—It has already been stated that the essential condition in this disease is the tendency to hemorrhage. The earliest hemorrhages are usually the subperiosteal ones and these are more frequently noted in the lower than in the upper extremities. They usually occur toward the epiphyses of the long bones, in the neighborhood of the knees and ankles. Other bones may be affected, however, notably the ribs and the bones of the arms. German authorities consider a backward displacement (dislocation) of the sternum and costal cartilages as

well-nigh pathognomonic; but this can only take place in advanced cases. Naegeli, Schoedel, Nauwerk, Schmorl, and Fränkel (quoted by von Starck) describe typical changes in the bone marrow and consider them essential. Separations between diaphysis and epiphysis have occurred. Hemorrhages may occur in a great many situations, viz.: The brain membranes, the tissues in the neighborhood of the orbit, the retina itself, the nasal mucous membrane, the gums, the lungs, the stomach or bowels, the kidney, the scrotum, the skin, etc. Aside from this hemorrhagic tendency, the only other pathologic find is the anemia.

The red blood-corpuscles have varied in the observations we have made from 2,200,000 to 3,800,000 in a cubic millimeter, hemoglobin, from 80 per cent. to 50 per cent. The red blood-corpuscles frequently presented their regular appearance known as poikilocytosis, with no other notable change. In one case slight pigmentation was observed. Slight leukocytosis is also seen with a pronounced preponderance of the mononuclear forms.

**Symptoms.**—The symptoms of infantile scorbutus are both constitutional and local. Rarely the attack may be precipitated by a number of acute symptoms, gastro-intestinal in character, with fever and the constitutional disturbance associated therewith. The temperature ranges from 100° to 102° F. (37.8° to 38.9° C.); a fever of 105° F. (40.5° C.) and above is rare. More often the disease develops insidiously. The child becomes peevish and fretful, the appetite capricious, though usually poor, temperature but slightly above normal, most frequently holding within the normal range. The child lies upon its back, with limbs extended or slightly flexed. The anemia, which at first was slight, becomes more marked, and with it excessive irritability. Pressure along the tibia and femur and about the knee and ankle-joints is attended with considerable pain; the child cries out, but makes but feeble effort to be released. If the disease is far advanced, the position becomes characteristic, the pain becomes acute—now the slightest movement causes the little patient to cry out as if in great suffering. The excessive tenderness about the extremities has frequently occasioned the affection to be mistaken for one of acute rheumatism. If the skin is carefully examined, not infrequently distinct petechiæ or ecchymoses may be observed upon the legs and thighs. Oftener the mucous membranes, especially the gums, show evidences of the disease; though there may be but slight swelling. This is frequently the case before the eruption of the first teeth; afterward the gums become swollen, red, and suffused with blood. The spongy areas show a tendency to

bleed upon pressure. This condition may grow worse, going on to ulceration, and presenting in places patches of localized gangrene, the overlapping of the gums completely obscuring the teeth from view. In a case which came under our observation which had been treated locally with a solution of nitrate of silver there was presented all the appearance of linear ulcers along both the upper and lower gums, with exuberant granulations from which there was constant oozing of blood. In Cheadle's first patient the country physician had been snipping away the exuberant granulation tissue from the gums day by day. A bleeding mass protruded from between the child's lips. The joints themselves are seldom affected. The swelling is along the shaft of the long bones, about the line of separation between the diaphyses and epiphyses, and outside of the joint proper. The parts are sensitive to pressure, and become extremely painful as the disease advances. There is no rise in the local temperature of the part. The indisposition of the patient to move the limbs is due, primarily, to the pain which is produced upon motion, and in advanced stages of the disease to muscular weakness. This symptom has frequently been mistaken for paralysis. The electric reactions of the muscles are, however, normal. Usually the knee-jerk is not lessened. This condition of pseudoparalysis is more rarely present in the arms.

*The heart and circulatory apparatus* present no symptoms of importance, except partaking of the constitutional symptoms associated with the febrile movement. In a number of instances hemic murmurs have been noted where the anemia has been marked. The presence of moist rales in the lungs, posteriorly, is not unusual. Hemoptysis is rare, but when present, may be considered a grave symptom (hemorrhage into the lung).

*Kidneys.*—Hematuria is not infrequent. The presence of hyaline and blood-casts is not rare, according to various authorities. In nine cases observed by us, however, in but one were blood-casts and hyaline tube-casts noted.

The symptoms of infantile scorbutus vary in degree from attacks of slight severity to the more advanced and aggravated forms. We have observed that the hemorrhagic condition of the gums is no guide to the state of the lesions in other parts of the body. For instance, in a case which presented but slight swelling of the gums there were persistent hematuria and numerous spots upon the skin. On the other hand, in a case in which the gums were extensively involved, the swelling about the joints was slight, with no marked symptoms referable to the lower extremities. In another, where the patient was brought to us as a

supposed case of rheumatism, the weakness in the lower extremities was considerable, the skin showing a marked petechial eruption over the anterior portion of both legs, with no involvement of the gums; and in still another, the hemorrhagic state was general: on the gums, about the pillars of the fauces, with the presence of blood in the urine and stools, and no involvement of the skin. In a case supposed to be one of infantile palsy there was simply weakness in the lower extremities, extreme sensitiveness to pressure over the shafts of the tibia and femur, with slight sponginess of the gums, marked diarrhea, and profound anemia. Hemorrhage has rarely taken place into the brain membrane (pachymeningitis hemorrhagica interna) with resulting meningeal symptoms. In the neighborhood of the eye it has caused discoloration and exophthalmos (causing a suspicion of malignant growth). Epistaxis is not uncommon. In rare instances the resulting tumefactions from subperiosteal hemorrhages of the extremities have given rise to the diagnosis of multiple sarcomata.

**Diagnosis.**—From the insidious nature of infantile scorbutus, the history of the case, and the character of the symptoms there should be no difficulty in reaching a correct diagnosis. General debility, anemia, sponginess and bloody extravasation of the gums, petechiæ and ecchymoses upon the skin, especially upon the lower extremities, enlargement and tenderness about the joints and along the shafts of the long bones, and an apparent loss of power, muscular rather than nervous in origin, in infants fed upon one of the proprietary foods or sterilized milk, present a picture characteristic of scurvy. Finally, the therapeutic test referred to under treatment, clinches the diagnosis.

Until within recent years infantile scorbutus was considered a rare affection, doubtless due to the old classification, which included the disease under the head of rickets, purpura in its various forms, and hereditary syphilis.

**Differential Diagnosis.**—The first essential for the diagnosis of infantile scurvy is a knowledge of its existence and characteristics in early life. Few cases, when properly diagnosticated and treated, redound more to the credit of the medical attendant or consultant. Infantile scorbutus is most frequently confounded in the early stages with acute rheumatism. The records of cases show this to be a most common source of error. J P. Crozer-Griffith rendered a signal service to pediatrics when he entitled his classic monograph "*Not Rheumatism, but Scurvy.*" Rheumatism in infancy is a very rare disease. From rickets scorbutus is to be differentiated by the

history of the attack and absence of the evidences of rickets, also the rapid subsidence of the symptoms under treatment. In those cases where scurvy occurs in children previously the subject of rickets the diagnosis might appear difficult; but even here the rapid disappearance of the acute symptoms under treatment would aid us in eliminating a distinctly constitutional disease. Syphilis too must be recognized by its own characteristic signs. It affects the same regions of the long bones that scurvy does, but syphilitic epiphysitis usually occurs within the first two months of life, scurvy almost never. The Wassermann test will greatly aid in obscure cases.

**Prognosis.**—Except in the advanced stage of the disease, associated with marked constitutional disturbance, the prognosis is good. Fortunately, it is a disease which rapidly responds to treatment. In those cases seen late, and which have been neglected, the vitality being low, the hopes for recovery are not encouraging, though even here it is astonishing what can be done by a properly arranged regimen. It is probable that the disease still has a large mortality because of the common failure to make a correct diagnosis.

**Duration.**—The duration of the disease is variable. In cases early placed under treatment improvement may be rapid, the acute symptoms rapidly subsiding. Instances where the disease extends over a longer period—from six months to a year—are rare; the danger of laying the foundation for organic disease must not be lost sight of. Relapses are not uncommon.

**Treatment.**—The etiology of this affection is a sufficient guide to the treatment. First, change of food to a diet rich in fresh foods is all essential, and as a *sine qua non* of a character suitable to the age of the child. Orange-juice, beef-juice, with the use of perfectly fresh clean milk, for infants, have proved ample in our hands. Medicinally, the use of minute doses of citrate of iron and, later, arsenic in the form of Fowler's solution may become useful adjuncts, but they are seldom needed.

The baby should be kept in a supine position on a firm hair pillow, even bandaged to it, and the extremities should be fixed upon well-padded splints until the pain and tenderness have completely disappeared. Better than these, we consider a light frame made from three barrel staves bound together. We pad this frame with cotton, and bandage the patient on it. He may thus be carried, even into the open, without handling his tender extremities. Handling of the parts should be very gentle for many weeks subsequent to convalescence.

## SIMPLE ATROPHY

**Synonyms.**—INFANTILE ATROPHY; MARASMUS; ATHREPSIA; SIMPLE WASTING

A pathologic condition characterized by extreme wasting, common enough as the result of many forms of disease in infants and young children, especially the subacute or chronic gastro-intestinal diseases. The majority of cases of so-called marasmus are nothing more than continued starvation, produced by the lack of nutritive elements in the food, supplemented by a chronic toxemia from milk bacteria, producing first a continued gastro-enteric catarrh, and later the group of symptoms of which wasting is the most prominent symptom. It is doubtful whether infantile atrophy ought ever to be described as a separate disease—it is rather a group of symptoms—and yet many authors of wide experience, notably Eustace Smith, Holt, and Starr, have so described it. Holt defines infantile atrophy, or marasmus, as “the extreme form of malnutrition seen in infancy, occurring, so far as is known, without constitutional or local organic disease. It is a vice of nutrition only.” It must be clearly borne in mind that the immediate cause of true simple atrophy lies more in the weak assimilative powers, inherited or acquired, of the infant itself, than in any fault of its food, although the latter may be a potent predisposing factor. All diseases in which wasting is a symptom, such as tuberculosis, infantile syphilis, and diseases of the stomach and intestines, must be excluded from this class.

**Etiology.**—Marasmus is seen much more frequently among the poor—the class who come to hospitals and dispensaries—than among the well-to-do. It is a disease of the city slums, where children are crowded in tenements amid the worst hygienic surroundings. When seen among the better classes it is usually in infants born prematurely or in children of parents who for generations have been under the average of health or the victims of inherited disease. It occurs in premature infants not infrequently, and especially in premature children of very young, badly nourished mothers. By far the largest number of cases which come under our notice have been fed on badly prepared milk, usually milk and water, none too clean, the proprietary foods, and condensed milk. “Table food” given too soon may be a cause. Occasionally, but rarely, we have seen a milder form of marasmus in infants whose mother’s milk was shown, by analysis, to contain all the elements in proper proportion, but in all of these cases the children themselves were

below the average in weight and general health, and in some of them evidences of rickets later appeared.

Many theories have been advanced to explain the occurrence of atrophy, particularly of the rapid forms that occur in the early months of life. Two recent theories, those of Wentworth and Edsall are worthy of more than passing attention, for they really seem to explain. The former performed secretin experiments upon cats. He found that scrapings from the duodenums of atrophic infants were deficient in secretin, as manifested in his experiments upon cats. (The pancreatic



FIG. 38.—ACUTE ATROPHY (ACUTE MARASMUS).—(From patient in the Department of Obstetrics and Diseases of Infancy, Polyclinic Hospital, Philadelphia.)

response, when a glycerin emulsion of the scrapings was injected, was delayed or absent.) He also found hypoacidity quite constantly. Edsall, working independently, had published the results of his erepsin experiments prior to Wentworth's publication. He found erepsin deficient in the intestinal mucosæ of atrophic infants. He concludes that there is either a failure of proper proteid disintegration (into amido- or amino-acids) during absorption, or else that the proper reconstruction into tissue proteids does not take place. It seems to us that the work of these two investigators, considered jointly, furnishes us with more than a tenable hypothesis in explanation of infantile atrophy. Practically they have apparently shown that failures of the secretory and absorptive functions are responsible for the condition.

Holt summarizes the etiology of the disease as a failure of assimilation from imperfect digestion, due to improper food and unhygienic surroundings or feeble constitution, and from this results a progressive

loss of weight, feeble circulation, imperfect expansion of the lungs, and, in consequence of this, deficient oxidation of the blood takes place. The temperature is lowered, and finally a deterioration occurs in the blood itself. At last a point is reached where the small amount of resistance ends and the child dies.

**Pathology.**—Autopsies made on children dying of marasmus are generally unsatisfactory in their results, so far as finding any changes pathognomonic of the condition. Holt states that in one-third of his autopsies he found fatty degeneration of the liver; the organ was enlarged and considerably above the normal weight.

The brain is usually anemic, with dark fluid in the sinuses; marantic thrombi are rare. Frequently small areas of hypostatic pneumonia will be found in the lungs; these are most common on the posterior borders of both lungs, involving the pulmonary tissue to a depth of half an inch, but are probably secondary. Areas of atelectasis may be seen in the lower lobes of the lungs of young infants; the pleuræ are normal. The heart, spleen, and kidneys are anemic, but otherwise normal. Dilatation of the stomach is sometimes present, and atrophy of its mucosa has been noted. The intestines contain food and sometimes mucus. Some enlargement of the solitary follicles of the colon and small intestine may occur, Peyer's patches may be increased in size, and enlargement of the mesenteric glands may also be seen.



FIG. 39.—ATROPHY OF AN EXTREME DEGREE IN A SIX-MONTH-OLD BABY.—(*Philadelphia Polyclinic Hospital.*)

**Symptoms.**—At birth such children may be fairly well nourished and may so continue until for some cause weaning is necessary, and the infant is fed on some infant food or badly prepared milk. From this time the child begins to lose weight, and although it may be fed frequently, it seems to have a constant desire for food. Almost from the first the infant is irritable, crying continually and never seeming satisfied. Soon the loss of weight begins to show: the form loses its

plumpness, the outlines of the ribs are seen, and the various joints are plainly marked. The wasting is most distinct in the limbs and face, the latter assuming an expression which is eminently characteristic of the disease. The eyes become sunken, and the great wasting in the lower part of the face makes the forehead, by contrast, appear unusually prominent. The whole face assumes a triangular shape, the ears are prominent, the cheeks are sunken, while deep furrows appear around the mouth. The buccinator pad of fat remains. The expression is curiously aged. The child soon becomes anemic, with considerable reduction in hemoglobin. Holt states that in his cases he has seen the hemoglobin as low as 30 per cent., and in one case it was reduced to 18 per cent. There is extreme pallor, especially of the face, except late in the disease, when patches of pulmonary atelectasis occur, giving the skin, from nonaëration of the blood, a somewhat leaden hue. Heart murmurs, due to anemia, are frequent. The abdomen is generally distended and filled with gases, produced by faulty digestion. The muscles appear to be small and atrophic, and are covered by no fat. The skin hangs in folds, and is generally dry and scurfy. Various forms of skin eruptions are common; the buttocks and genitals are covered by erythema. Thrush or other forms of stomatitis may appear; bed-sores appear later. The temperature may be normal, subnormal, or slightly elevated. Most commonly it is below the normal; it may fall as low as 95° or 96° F. (35° to 35.6° C.). Not infrequently there is a slight rise at night. The appetite, which is generally ravenous in the early part of the disease, fails entirely, and when food is taken, it is followed by severe vomiting. The tongue is coated, the digestion poor, and the power of assimilation in most cases is practically *nil*. The bowels are constipated or irregular; there may be diarrhea. The movements are greenish-yellow in color and contain considerable mucus. Colic is nearly always present. The nervous symptoms are sometimes very severe; in the earlier stages the child is restless, particularly at night; the whole nervous system is hypersensitive, this showing itself in a strong tendency to convulsions. In the late stages there may be some retraction of the head and a decubitus much like that seen in tubercular meningitis. The cry of the advanced case is characteristic. The face is wrinkled and it is clearly apparent that the child is crying, but there are no tears, and often no sound is heard. This type of cry is an ominous phenomenon. Physical signs referable to the heart and lungs are generally negative, except that there may be anemic murmurs, before referred to, and a certain amount of bronchial irritation is very commonly seen. Small

areas of pulmonary atelectasis may be found, or the evidences of hypostatic pneumonia; these latter usually appear toward the end, and are of grave import. The urine is of a dark yellowish color, and ranges from 1010 to 1013 in specific gravity. Albumin and, more rarely, sugar may be found in it. Edema may be a symptom, and when it appears, is nearly always a sign of a fatal termination. Purpuric blotches on the abdomen are likewise of bad omen.

**Diagnosis.**—The disease with which simple atrophy is most likely to be confounded is general tuberculosis; indeed, the symptoms of the two diseases are so much alike that it is impossible sometimes to differentiate between them without an autopsy. Cases of tuberculosis are sometimes seen with few symptoms except those of marasmus, and occasionally children apparently dying of simple atrophy will have a few patches of tubercle in their lungs. A careful physical examination that takes into particular consideration adenopathies, manubrial or stripe-dullness and pulmonary involvements, supplemented by the modern cutaneous tests for tuberculosis, will render us good service. Even when pulmonary dullness is present—and it often is late in the disease—it may be from hypostatic pneumonia. Holt, however, has pointed out that the dullness of hypostatic pneumonia is most likely to be found in the posterior portions of the lungs, while that of tuberculosis is commonly in the anterior *areas*. In some cases the infant affected with marasmus may be healthy at birth, the disease developing later, while tuberculosis may develop at a very early age. In tuberculosis the elevation of temperature is greater and more regular, with morning remission and evening rise. Frequently the pulmonary symptoms are rather more marked than are those of acute atrophy. Tubercular meningitis may be differentiated by the presence of general meningeal symptoms, the palsies, or contractures, the long-continued decubitus, and the full pulsating fontanel. The *fontanel* in simple atrophy is depressed. The scaphoid abdomen is usually seen in meningitis, while in atrophy it is enlarged or entirely depressed. In meningitis there is usually the hydrocephalic cry. Syphilis may be distinguished by its characteristic eruption, the presence of mucous patches, and the specific coryza seen soon after birth. Enlargement of the liver, spleen, and knee-joints will aid in the diagnosis.

**Prognosis.**—The outlook for recovery depends considerably on the age of the child, the younger the patient, the worse the prognosis. The duration of the disease is also a factor to be taken into account; naturally, a child affected with marasmus for a long time will be more

reduced in its powers of resistance than one in whom the condition has lasted but a short time. The most important element in the prognosis is the previous care which the infant has received, especially in the matter of its food and general hygiene. In those infants who have been fed for a long time on badly prepared artificial foods the prognosis is not the best. In our experience the worst cases have been those who have been fed on condensed milk since birth. In these the prognosis has generally been unfavorable.

**Treatment.**—The most important element in the treatment of simple atrophy is its prevention. This among the poor, the class in which it is most common, should consist in teaching the mothers from the first the proper manner in which to feed their babies. In most cases it is very little if any more trouble for a mother to prepare milk in a proper and cleanly manner than in an improper and dirty way, and she will usually do the former if she is taught how. In dispensaries a few clearly printed rules can be used, giving explicit directions as to the proper manner of preparing milk and other foods employed; these directions should include the general care of the child, as to its bathing, clothes, and exercise. These children should receive as much fresh air as possible, and the necessity of regular bathing should be impressed on the mother. It is also of importance that infants with weak digestive powers should be fed from the breast for at least nine months, providing the mother's milk is fairly good. If the maternal supply has become so much decreased that the infant cannot be entirely fed from the breast, it is better to use mixed feeding—partly breast and partly modified cow's milk—than wholly to depend on artificial feeding. Wet-nurses are of great use in this class of patients, and should be frequently employed if the infant is under six months old. Among the poor and ignorant the use of milk modified at home will often result in failure, yet by patiently teaching the mother how to feed her child we may often accomplish much. In hospitals and among the well-to-do artificial feeding by carefully prepared modified milk will frequently be successful, particularly if the infant is over six months old. At first a milk mixture low in proteids and fats should be used, and this will frequently have to be predigested. A formula such as the following may be found useful:

Fat .....	1	to 2 per cent.
Sugar .....	6	to 7 per cent.
Proteids .....	0.75	to 1 per cent.

If these are assimilated, the proportion of fat and proteids must be raised from time to time. If formula feeding does not succeed in these cases, we quite agree with Baginsky and others that buttermilk is practically a specific in infantile atrophy. The physician should also have other substitute foods at his command, not considering them ideal but simply as bridges which may convey the little patient from the danger region to one of comparative safety. The child should be weighed at regular frequent intervals. In very young infants with subnormal temperature incubation is often of use, or, a couveuse not being at hand, the child may be wrapped in raw cotton and laid in a basket or small crib, with one or two bottles of hot water or a hot-water bag. The management in these cases is very much the same as that of premature infants. Holt suggests that they be made to cry vigorously several times a day, in order to keep the lungs expanded. In older children massage with oil inunctions is of use. Drugs, as a rule, are of very little account. If the child improves, a change of air at the seaside or mountains may prove of benefit.

## DISORDERS OF METABOLISM DEPENDENT UPON THE ORGANIC CONSTITUENTS OF FOOD

### FOOD INTOXICATIONS—(RUHRÄH)

There exists a latter-day tendency to minimize the influence of intestinal bacteria, and to accentuate the disturbing influences upon metabolism of the sugars, fats and proteins of foods. Finklestein, Czerny, Kellar, Feer and others have contributed much to our knowledge of such metabolic disturbances. Ruhräh, in America, has made an important contribution to this subject. As yet, however, our conceptions are far from clear, and we believe that the pendulum of opinion has swung too far in this metabolic direction, and that it will inevitably swing back to a belief in the greater importance of endogenous and exogenous infections.

We shall quote briefly the views of Ruhräh:

**Symptoms.**—Periodicity of attacks is one of the most characteristic features. Such attacks vary greatly in their characters: vomiting (as in cyclic vomiting), headaches, fever, diarrhea, asthma and other symptoms are noted in the various attacks.

**Diagnosis.**—Ruhräh contends that a study of the child's habits and of his diet, when coupled with a careful physical examination (to exclude diseases of the various organs), will render the cause of the disturbance clear in more than half of the cases.

**"TOO MUCH FOOD OF ALL KINDS.**—This usually causes such attacks as are called biliousness. There is fever, a coated tongue, foul breath, headache, malaise, and often drowsiness. There is often vomiting, or diarrhea, or both. The liver may be somewhat enlarged and tender.

**"TOO MUCH PROTEIN.**—The symptoms are as in the preceding. Sometimes one symptom is especially prominent, as recurring headaches or attacks of vomiting, or in the milder cases, periods when the tongue is furred and the breath foul without much other disturbance." [Illman has reported some interesting cases in adults.]

**"TOO MUCH FAT.**—The child's general health is poor, the skin is pale and muddy, there are dark circles under the eyes, the breath is exceedingly fetid; there is frequently gastric disturbance and vomiting, and there is often diarrhea with the passage of undigested fat in the stools. [We have already dwelt upon the importance of fat constipation with dry, light colored stools, containing much soap.]†

**TOO MUCH CARBOHYDRATE.**—This is the most frequent form, owing to the fact that many children are given large quantities of starches and sugars. Recurring attacks of vomiting, diarrhea with fever, often headache, or asthma, are the most frequent symptoms.

**Prognosis.**—When the cooperation of the parent can be secured, the results are usually satisfactory.

**Treatment.**—The intestinal tract should be cleaned out with a brisk purge and occasional doses of phosphate of soda given. The diet should be carefully regulated to suit the child's age and condition. When any special class of foods is at fault, it should be reduced to a minimum.

## DIABETES MELLITUS

Diabetes mellitus, also called saccharine diabetes or glycosuria, is a constitutional disorder of the elaborative functions of nutrition, characterized by a persistent and excessive secretion of saccharine urine, polyuria, great thirst, excessive appetite, rapid emaciation, and especially in children an early fatal termination. It is a rare disease in early life, most statistics placing its occurrence in childhood at from 1/2 to 1 per cent. of cases. Von Noorden believes that if urinary examinations were made more frequently in infancy and childhood, it would be found more common.

† *Note:*—According to Southworth, the "ammoniacal diaper" is another evidence of excessive fat.

**Etiology.**—Heredity, and especially an inherited lessened capacity for the digestion of the carbohydrates, seems to play a large rôle in its etiology. The disease is apt to be a legacy from a neurotic or gouty ancestry. Pavy and von Noorden have both recorded family histories presenting three successive generations of diabetics. Long-continued dietetic errors, by deranging the process of nutrition, may predispose to it. Exposure to cold, traumatism, acute infectious diseases, climate, syphilis, and malaria, disorders of the liver and pancreas, have all seemed to assume an etiologic relation. Its frequency among Jews is remarked by all writers, though this is believed to be less often observed in childhood than in later life (von Noorden).

**Morbid Anatomy.**—No constant lesion is present in diabetes; those encountered other than pancreatic lesions, are usually consequences, rather than causes, of the disease. The pancreas has been found to be the seat of disease in a large proportion of cases, the conditions observed occasionally being atrophy, fatty degeneration, supuration, and fibrous inflammation, concretions, cysts, and tumors of this organ. Such definite conditions have not been found always in childhood, but extreme smallness of the organ has been noted. Complete extirpation of the pancreas in animals always produces diabetes, while partial removal or ligation of the duct is not followed by such a result (von Mehring and Minkowski). These experiments suggest that it is one of the functions of the pancreas to control metamorphosis of sugar in the body. It has been suggested (Lepine) that the pancreas produces a ferment which is necessary to the normal metamorphosis of sugar. The liver is often enlarged and fatty; cirrhosis and pigmentary degeneration have been observed. The lungs often present evidences of tuberculosis but these are secondary in character. The kidneys in many instances are diseased.

The infrequency of diabetes in childhood is to be explained by the activity of the nutritive processes in early life. No theory, physiologic, pathologic, or chemic, will explain all cases of diabetes. It is known that it sometimes follows diseases and traumatisms of the central and peripheral nervous systems. Again glycosuria or true diabetes may follow infectious diseases.

**Symptoms.**—The disease in children differs from its course in adults chiefly in its more rapid, often sudden, development, and in its early fatal termination. Von Noorden differs from this commonly accepted view to this extent. He thinks that a mild glycosuria has usually persisted for some time before the acute symptomatology is

manifested. It is not in reality an acute onset, but rather a rapid transition into a grave malady. The child, in spite of excellent appetite and ample food, grows thin and emaciates rapidly, and the skin becomes dry and toneless. Thirst and appetite are excessive. There is a frequent desire to void urine, which is passed in large quantities and upon examination is found to be usually of high specific gravity, markedly acid, pale and sometimes greenish in color, sweetish of taste, aromatic in odor, and to contain sugar and to ferment rapidly. The amount of urine may vary from 1000 to 6000 c.c. (1 to 6 qts.) in twenty-four hours, the specific gravity being from 1030 to 1040, the proportion of sugar from 1 to 10 per cent. If acetone, diacetic acid and oxybutyric acid are present in the urine diabetic coma is imminent. Incontinence of urine is often the first symptom; and when accompanied by thirst and marked wasting is always suspicious. Peevishness, restlessness, itching of the genitalia, constipation, and sensitiveness to cold are other symptoms frequently observed. The tongue is beefy, red, and may be fissured, the teeth bad, the skin dry and roughened, and the patellar reflexes are diminished and at times lost.

**Diagnosis.**—A recognition of the persistent presence of sugar in the urine determines the diagnosis. A careful examination of the urine should be made in all cases of polyuria or incontinence. True diabetes must be distinguished from transient glycosuria, however, which is of occasional occurrence in infancy and childhood. It may follow excessive indulgence in saccharine food or may be noted in the wake of an infectious disease. Von Noorden believes that the toxin of diphtheria, etc., may exert a malign influence upon the pancreas. It is rare for such a glycosuria to persist more than a few days. Whenever possible, it is desirable to examine a portion of the entire quantity of urine passed in twenty-four hours—where this is not obtainable, a specimen voided two to four hours after a meal is most likely to show the presence of sugar when the amount is small.

**Fehling's Test.**—For *qualitative* testing take one cubic centimeter of the test solution and dilute it with four cubic centimeters of water; boil this, and if no precipitate occurs and the solution remains clear, it is fit to be used; if not, a fresh solution should be obtained. To the test solution, after boiling, add the urine, drop by drop, until a bulk not exceeding the amount of test solution has been added; if no yellow or red precipitate takes place, sugar is absent.

An approximately accurate *quantitative* test can be made with Fehling's solution, employed as in the foregoing test, if it is remembered that an equal amount of urine which exactly reduces the test solution

contains  $1/2$  of 1 per cent. of sugar. If the color is removed by an amount of urine equal to half of the bulk of the test solution, that urine contains 1 per cent. of sugar; if the amount of urine necessary to remove the color from the test solution amounts to twice the bulk of the test solution, it contains  $1/4$  of 1 per cent. Urine containing a large percentage of sugar should be diluted in the proportion of 1 to 9 of water, and the diluted urine employed in testing for sugar; the result obtained should be multiplied by ten.

*Phenyl Hydrazin Test.*—To fifty cubic centimeters of urine add from one to two grams of hydrochlorate of phenyl hydrazin and two grams of sodium acetate; heat on a water-bath one hour; on cooling there will appear at the bottom of the beaker a crystalline or amorphous precipitate, which under the microscope has the form of the characteristic yellow needles of phenyl glucosazone. It is claimed that this test will show 0.05 per cent. of sugar. It gives no reaction with the other organic substances in the urine, as uric acid, kreatinin, hippuric acid, etc.

*The Fermentation Test.*—This serves the double purpose of a quantitative and a qualitative test. Fill a four-ounce bottle with the urine whose specific gravity has been determined. To it add a piece of compressed yeast, the size of a bean, or a teaspoonful of brewer's yeast; mix thoroughly, and stand in a warm place ( $70^{\circ}$  to  $80^{\circ}$  F.— $21.1^{\circ}$  to  $26.7^{\circ}$  C.) for twelve hours or longer. At the expiration of this time the sugar will have been converted by fermentation into carbonic acid gas and alcohol and the specific gravity lowered. For every degree lost in specific gravity there is one grain of sugar to the fluidounce. Thus, if the original specific gravity is 1040, and after fermentation 1020, there are twenty grains of sugar to the fluidounce. From this the percentage may be ascertained by multiplying the number of degrees lost by 0.23. Thus, in urine losing twenty points in specific gravity the percentage of sugar would be 4.6 per cent.

**Prognosis.**—Diabetes in children is essentially an incurable disease; the younger the patient, the more rapid the fatality. The course of the disease rarely exceeds six months. Rarely, according to Van Noorden, mild cases may be observed. Thus he knows one patient who first exhibited glycosuria at seven years of age. He has never since been able to ingest more than a small amount of carbohydrate food. He now has a son who is thirty years old, but who has exhibited the same tendency to glycosuria since he was a child of four.

**Treatment.**—In children diabetes does not permit of classification as it does in adults, in whom we recognize at least two forms of the

disease. Few recoveries from true diabetes in children have been reported. Dietetic treatment is most important. We do not expect it to save, but it certainly does prolong life and render it more comfortable. Cane sugar is forbidden; but it is unwise to shut off from the carbohydrate element. The oat-meal treatment is certainly worthy of trial, as it seems to furnish a harmless form of carbohydrate food. The danger of excluding the starches entirely is the danger of acid intoxication. Diets should be arranged on a calorimetric basis. In one personal communication, however, we have received information that seemed to show a recovery in a boy of five years who was treated by injections of glycerin emulsion of the pig's pancreas. The glycosuria had followed an infection, however, and the case may, therefore, be open to question. The hygienic treatment is important, especially the maintenance of the best condition of the skin. A proper amount of exercise helps to use up the available carbohydrate in the economy, and is to be recommended. If the patient is too weak, passive exercise and hydrotherapy may supplant it. Alkalies are of value in threatened coma.

### URIC ACID AND URIC ACID CONDITIONS

At the present time there are divergent views as to the source of uric acid and the seat of its formation, although the trend of opinion seems to be in favor of the view that assigns to the kidneys the office not only of secretion, but of actual elaboration. This belief holds that uric acid is derived from the nuclein of the body-cells (chiefly the leukocytes) and not directly from the proteid elements of the food, except as the latter promotes a digestive leukocytosis, thereby increasing the destruction of nuclein. Urea also may contribute its share.

Uric acid is *increased* by food rich in nuclein—bone-marrow, liver, brains, pancreas, veal, tea, coffee, meat extracts, and asparagus—and in any disease that impairs respiration or circulation, in affections of the liver and spleen, in anemia, in most acute diseases, and in gout following the paroxysm; also by certain drugs, as pilocarpin, salicylic acid, antipyrin, phosphorus, etc. It is *decreased* by non-nitrogenous diet, in advanced stages of disease of the kidneys, in gout during the paroxysm, and in most chronic states.

Uric acid is normally in the urine in the form of what is known as the mixed urates of sodium, potassium, and ammonium (acid and neutral urates). The neutral salts are freely soluble in water, the acid urates feebly so, while uric acid itself is almost insoluble. The

uric acid and urates are held in solution by the presence in the urine of the normal coloring-matters, by the salts (chiefly the chlorids), and by a low degree of acidity; for with a reduction in the proportion of the salts and pigments and an increase in acidity the uric acid will be precipitated, although there may be less than the normal amount present. This precipitation, however, is pathologic only when it takes place while the urine is still within the body. It must be remembered that the harmful effects of uric acid are due solely to the mechanical irritation of its crystals, whether they be precipitated within the urinary tract or in the tissues of the body, for uric acid itself possesses no poisonous properties.

When crystals of uric acid are deposited in the tubules of the kidney, there may be present the symptoms of renal irritation—sense of weight or pain in the lumbar region, or pain referred to the umbilicus; occasionally there may be nausea and vomiting. In other cases the child may only show signs of mild mental depression, irritability of temper, and insomnia, with variable appetite; constipation is also apt to be present. Sometimes enuresis may be the only symptom. Whenever such a train of symptoms, without obvious cause, makes its appearance suddenly, suspicion should always point to a possible uric acid precipitation and suggest an examination of the urine.

When the tubules of the kidneys are the seat of actual irritation from the continued presence of uric acid crystals, an examination of the urine would show the presence of small amounts of nucleo-albumin and serum albumin, usually hyaline casts and cylindroids, and, occasionally, a few granular and epithelial casts, or even leukocytes and erythrocytes. No doubt many cases of granular kidney have had their origin in this continued irritation from uric acid crystals which has escaped recognition. Gravel and calculus are attended with symptoms identical with those in the adult.

Although few cases of gout in children are reported, it is likely that it is often not recognized.

**Treatment.**—The treatment is dependent upon whether the condition is one of excessive formation of uric acid or of a state of lessened solubility of the urine.

As a general rule meats should be allowed but sparingly, and only when a proper amount of exercise is taken. Thus exercise should always be insisted upon. Milk, white of egg, cereals, lettuce, celery, and fresh fruits constitute the best diet. Asparagus is to be avoided, as it contains one of the xanthin bodies. Fats and starches in moderation may be allowed unless they give rise to digestive disturbance.

When an oxaluria exists, spinach, rhubarb, tea, cocoa, coffee, tomatoes, strawberries, etc., are contraindicated, as they contain oxalate of calcium. Pastry and sweets have been shown by clinical experience to do harm.

The citrate of lithium or its carbonate as an effervescing draft is often useful. When the urine is strongly acid, alkalies should be employed, such as the citrate of potassium or bicarbonate of soda, in doses of from ten to twenty grains well diluted. An evening dose is most important, for it is during sleep in the long fasting period between the evening and morning meals that the urine attains its highest degree of acidity.

The urine must be examined immediately after being voided and preferably while still warm. For quantitative analysis, the method of Hopkins is preferred by the authors for ordinary use. When such test is impossible, an approximate estimation may be made in the following way: Given a specimen of urine in which uric acid crystals have been demonstrated by the microscope and in which there is a fair proportion of the normal coloring-matters and salts, accompanied with a moderate degree of acidity, it may be inferred that the precipitation is due to excessive formation.

## CHAPTER X

# DISEASES OF THE PERICARDIUM, HEART AND BLOOD- VESSELS

### GENERAL CONSIDERATIONS

**Anatomy.**—Normally, within the first ten days after birth the circulation may be said to lose its fetal type wholly and assume extra-uterine characteristics. The changes occur at slightly variable periods. They include the conversion of the ductus arteriosus and ductus venosus into fibrous cords; the closure of the foramen ovale; changes in the umbilical veins and umbilical arteries, the first forming the round ligament of the liver, the second the true anterior ligament of the bladder and the superior vesical arteries. (See Fetal Circulation.)

In the early weeks of postuterine existence one may clearly see the remnant of the Eustachian valve, and though the foramen ovale is usually closed, yet it is distinctly outlined. Variations in the weight of the heart at different ages are shown in the following table of Boyd's:

AGE	GRAMS
At birth.....	20.6
One and one-half years.....	44.5
Three years.....	60.2
Five and one-half years.....	72.8
Ten and one-half years.....	122.6
Seventeen years.....	233.7

The relative weight of the organ is greatest at birth, and the right side predominates to a slight degree over the left. In infancy and early childhood the long axis of the heart is more horizontal in the thoracic cavity than it is in later life.<sup>1</sup>

**Physiology.**—Independent of its nervous mechanisms the cardiac muscle appears to possess a property of rhythmic contractility. Much more stress has been laid upon this since the discovery of the bundle of His. The controlling influence of the nervous system is of extreme importance, however, in the lower animals. Ganglia and plexuses are found in the heart, the former being inhibitory or augmentory in function. The center for the extrinsic controlling mechanism lies in the bulb, inhibitory impulses passing from it down the pneumogastric

<sup>1</sup>Relatively, the heart is small and the blood-vessels very large in capacity. Thus, high arterial pressure is much less likely to result than in the adult.

nerves, and the majority of the augmenter impulses passing down the spinal cord and to the cardiac plexuses through the medium of the sympathetic system.

Inhibition is undoubtedly nature's method of conserving tissue and energy (anabolism), and the inhibitory centers are constantly active. Augmentation is synonymous with catabolism.

Though our knowledge of these central agencies is far from perfect, yet they cast many important side-lights upon the functional cardiac disturbances in children, and it seems clear that rational treatment must depend somewhat upon this physiologic knowledge. The average pulse-rate at birth is 136, and is usually somewhat faster in the female infant. For later periods of life Holt gives the following figures:

Six to twelve months.....	105 to 115 a minute.
Two to six years.....	90 to 105 a minute.
Seven to ten years.....	80 to 90 a minute.
Eleven to fourteen years.....	75 to 85 a minute.

Very wide variations from these average rates may result, however, from most trivial causes. (See Functional Disturbances of the Heart.)

**Examination of the Heart.**—The heart lies superficially in the young subject, but so rapid is the action, and so frequent are the variations in rhythm that a satisfactory examination may be extremely difficult. Sleep may furnish the best time to secure it in the infant or neurotic child. A careful pursuance of the various steps of inspection, palpation, percussion, and auscultation is always more or less fruitful of results.

The ribs in a child are placed more horizontally than in the adult; the diaphragm is higher, therefore the heart is higher in the thorax. The heart is also more horizontal, with the apical impulse, especially in the very young, felt somewhat outside of the mid-clavicular line.

The liver in young children is larger in proportion than in adults, hence, as the heart is in close contact with this, the area of cardiac dullness merges into that of the liver dullness below. These facts must be borne in mind in examining infants.

For a full description of the methods to be employed in the examination of the heart, the reader is referred to books upon physical diagnosis; but we shall briefly detail some of the observations that the careful student should make as he inspects, palpates, percusses, auscults and possibly measures.

**Inspection.**—Dwarfism or undue thinness of the extremities; exophthalmos, cyanosis; particularly of the lips and finger nails; dilatation of the veins, particularly those of the hands and of the

anterior surface of the thorax; throbbing of the great vessels in the neck; or the rarer venous pulse or diastolic collapse of the cervical veins; undue prominence (bulging) of the precordia; the visible apex beat and other areas where pulsation is observed (high on the thorax, epigastric, etc.).

**Palpation.**—Here remember that we are studying a circulation and not merely a heart. Begin with a study of the pulse, noting strength, volume, pressure, tension and quickness (of impact), etc., before resorting to the common study of pulse rate and rhythm. Nor should we rest content with a study of the radial pulse. The larger vessels may display important phenomena in a more exaggerated form. We quite agree with Sansom that palpation furnishes us with the most reliable method of locating the apex beat. Abnormal pulsations in other localities should also be noted.

**Percussion.**—Sansom states that percussion should not only inform us of the size of the heart, but also of the sizes of its different cavities. If one percusses with that thought in mind, he will glean much information. Use light percussion here, as in other such studies in childhood. Depend on the sense of resistance as much as upon the quality of the note.

**Auscultation.**—If one has followed the methods above outlined, one may know not only whether or not the heart is affected, but also in the former instance the character of the lesion; and this before he places his ear or stethoscope to the chest at all. Nevertheless, auscultation is very important. At this time, we shall not dwell upon the various things that may be heard, but shall rather content ourselves with outlining the method to be observed.

**Study.**—1. The sounds of the heart. Is the first sound at the apex strong? Which is the stronger, the systolic or the diastolic sound? 2. If the diastolic sound is the louder, even at the base, proceed to determine whether the aortic or the pulmonic second sound is more intense. 3. One is then prepared, and only then, to properly appreciate adventitious sounds (murmurs, etc.). What is the point of greatest intensity of the murmurs? In what directions are they transmitted? We much prefer the Bowle's stethoscope to any other form that we have employed.

We would not, however, recommend the student to depend *wholly* upon instruments which amplify sounds, but carefully to train his ear by studying cardiac sounds with the intervention only of the examining towel. Thus can the quality of sounds, their rhythm, force, and intensity, be fairly compared.

We can scarcely avoid mention of an old mode of examination to which Benedict has wisely redirected attention. We refer to a combination of the last two methods, or *auscultatory percussion*. This method has been frequently applied in outlining the gastric viscus, but not until lately have we been enabled to demonstrate its value in mapping out the size of the human heart. Only the *deep* or "*relative*" cardiac dullness can be studied thus, but this is the area which tells us most surely of the cardiac bulk and sizes of various cavities. The lightest immediate percussion will serve in this study. The outlines of the heart may be traced upon the chest with an anilin dye or some oily preparation. These lines can then be transferred to tissue-paper. In this way exact records may be secured and kept.

Starr prefers to inspect and auscultate before resorting to the more disturbing methods of physical examination.

The following aphorisms are drawn from Crandall:

1. The apex lies higher in the chest and further to the left than in the adult.
2. The apex-beat is hard to detect in the infant. In the child palpation shows this easier than in the adult.
3. The area of dullness is comparatively large. [Rotch indicates three stages in infancy and childhood during which differences are noted in relative and absolute dullness.]
4. Murmurs are heard over comparatively large areas. A study of differences in the quality of the sounds and points of greatest intensity will help us here.
5. The rate may be increased and the rhythm altered by slight causes.
6. In rachitic children and in those affected by empyema or pleural effusions and adhesions the apex may appear in an abnormal position.
7. Prominence of the precordia is sometimes marked. Normally the loudest sound is the first sound at the apex; the weakest sound is the second sound at the aortic cartilage.

The examination should always be performed during sleep, or in a state free from physical or psychic disturbance, and a child should never be frightened with a formidable looking stethoscope or other instrument. The use of the X-rays may often supplement these other methods of physical examination. In the hands of an expert most exact knowledge may be yielded. Blood-pressure studies have assumed so much importance in recent work that they will be dealt with in a separate chapter.

**The Importance of Physical Signs other than Murmurs in the Diagnosis of Valvular Disease of the Heart.**—Text-books teach that an endocardial murmur is not always an evidence of a valvular lesion, and also that a valvular defect may exist and still no murmur be present. Hamill and Le Boutellier and others have shown that inorganic murmurs are rather common in childhood, despite many statements to the contrary. They describe a late systolic murmur in a very detailed manner. It is usually best heard at the pulmonic cartilage; it does not obliterate the first sound but follows it early in the systolic interval; it is best heard at the end of expiration and is often obliterated by a full inspiration; it is accentuated by the recumbent posture and is sometimes only heard then. There may be a valvular or muscular disease without a distinct murmur being audible, and therefore other signs than murmur must be used in determining the existence of a valvular lesion. (See above.) Every valvular lesion of importance must result in hypertrophy and dilatation of the heart behind the valve diseased, and the evidence of such enlargement, in connection with some of the other finds just detailed must play important rôles in accurate diagnosis. We can almost say with Walsh that “the diagnosis of cardiac disease by cardiac murmurs is a relic of a by-gone century.”

Hypertrophy may be recognized by the heaving, forcible apex impulse, though one may be misled by a thin chest-wall. Epigastric pulsation may call attention to enlarged right heart. The jugular pulse, the hepatic, and the capillary pulse are all of diagnostic value. The visible pulse of aortic regurgitation is almost pathognomonic.

Palpation is very important, certainly *most* important in locating the apex-beat. Characteristic thrills may be perceived by palpation. The force of the cardiac impulse and the sites of abnormal pulsation are also learned through touch. Extracardiac causes for murmurs, such as might arise in a heart dislocated by pressure or retraction, can usually be excluded by careful percussion.

A weak aortic sound may be an indication of obstruction. The reduplicated second sound may point to valvular disease (stenosis of mitral orifice). A sharply accentuated first sound at the apex is likewise common in mitral stenosis. The peripheral tones in aortic regurgitation are a valuable confirmation.

Error in calling an inorganic murmur organic is readily made unless the secondary sounds are carefully sought for. We must not undervalue the importance of endocardial murmurs, but bear in mind that it is only by the complexus of symptoms that an accurate diagnosis can be

made. Of all the evidences of heart disease, the least valuable is the endocardial murmur.

### CLASSIFICATION OF CARDIAC DISEASES AND DISEASES OF THE PERICARDIUM

Time of occurrence	Nature of the affection	Clinical disease
Intra-uterine existence or very early infancy.	Developmental or Inflammatory. Various motor or sensory phenomena, unaccompanied by sensible changes of structure.	Various congenital affections. Functional diseases of the heart.
Extra-uterine existence (infancy or childhood).	Organic { Mechanical. Inflammatory. Miscellaneous.	{ Dilatation } Alone or as accompaniment of inflammatory change. Hypertrophy } Pericarditis, acute or chronic. Endocarditis, acute or chronic. Myocarditis, acute or chronic. Effusions (non-inflammatory). Granulomata. Neoplasms. Fatty degeneration of the heart muscle.

### CONGENITAL DISEASES OF THE HEART

To the embryologist and the pathologist these lesions are of considerable interest. To the clinician less importance attaches to them, because exact diagnoses are sometimes impossible, and manifestly our best therapeutic measures are often utterly useless. In the last few years, however, a rather exceptional experience has led us to array ourselves with those who believe that not infrequently an accurate diagnosis is at least as possible as it is in the presence of acquired cardiac lesions.

We believe that congenital cardiac disease is much more common than it is usually supposed to be, and that as a cause of death in early infancy it is frequently unrecognized.

Hirst, following Baginsky, adopts the classification below appended:

1. *Patency of the Foramen Ovale*.—This lesion is of slight importance, unless accompanied by a marked defect of the muscular wall. As a rule, this lesion is not accompanied by a murmur, and if it is, the sound is likely to be diastolic in time. Holt found small openings in fully one-fourth of his autopsies on infants under six months old.

2. *Defect of the Ventricular Septum*.—Absence of this structure causes the *cor triloculare*. Sometimes the auricular septum is also

absent, and we have the *cor biloculare*. This lesion is commonly associated with pulmonary stenosis. When in the presence of congenital cardiac disease one hears a murmur which is systolic in time, whose greatest intensity is at the midsternum, and which is transmitted toward the left, the probability is that there is a defect in the septum ventriculorum. (See Fig. 40.) One of us saw a typical instance of *cor triloculare* in an autopsy at the Philadelphia Hospital.



FIG. 40.—Congenital cardiac disease with polycythemia and cyanosis. The right border of the heart (light percussion) extends far to the right of the sternum. The liver is also enlarged. A loud blowing systolic murmur is best heard at +.—(*Philadelphia General Hospital*.)

3. *Anomalies of the Auriculoventricular Valves*.—These are much more common on the right side of the heart. Such lesions may be inflammatory (sclerotic endocarditis) or contractions may follow at the sites of small hematmata (Rotch). More rarely the valves are bound together, forming an annular diaphragm, and in exceptional cases atresia of the orifice may be found.

4. *Stenosis and Atresia of the Pulmonary Artery*.—Stenosis of this vessel is the most common and the most important congenital affection, for, unless some sudden strain be thrown upon the heart, patients may reach adult life. It exists most frequently in combination with a patulous ductus arteriosus and a deficient septum ventriculorum.

The lesion may be due to intra-uterine endocarditis or it may be developmental. Complete atresia is exceedingly rare. Obviously, the blood supply to the lungs would be materially restricted. Thus, one is not surprised to learn that many of these cases perish from pulmonary tuberculosis. Subsequent valvulitis is also to be expected. Stenosis of the conus arteriosus is occasionally observed. A diagnosis of pulmonary stenosis is based upon the symptoms and signs of congenital cardiac disease, and upon the following finds: (1) A systolic murmur heard best at the pulmonic cartilage, and not transmitted into the great vessels. (2) The presence of a decided thrill at the same site. (3) Right-sided hypertrophy is sometimes observed, though it need not be present to a great degree when there is a deficient septum ventriculorum.

5. *Persistence of the Ductus Arteriosus*.—Absence of this vessel has been observed. The obliterative endarteritis (Warren) by which this structure becomes closed may involve the isthmus aortæ. The murmur present in patulous ductus arteriosus is systolic in time, heard best at the pulmonic cartilage, but differs from that of pulmonary stenosis because it is transmitted along the great arterial trunks.

6. *Stenosis of the Aorta*.—Stenosis of the aorta is much less common than the similar condition of the pulmonary artery, and is inversely of more serious potentiality. Complete atresia may exist, and, as in deformities of the right side of the heart, the conus arteriosus may be stenosed.

7. *Transpositions of the Arterial Trunks*.—These cases, which may be observed alone or in connection with other visceral transpositions, rarely live to term. When other visceral transpositions accompany the cardiac one, less difficulty should be experienced in diagnosis.

8. *Numerical Anomalies of the Valve Segments*.

9. *Gross Anomalies*.—Acardia; ectopia cordis; displacement upward and downward; ill-developed heart; bifid apex; absence of pericardium, etc. These anomalies may accompany other gross developmental malformations, such as spina bifida or hydrocephalus.

**Symptoms**.—Cyanosis is present in about 90 per cent. of cases of congenital anomalies of the heart (Osler). It usually appears early; but in a case seen by us at the Philadelphia Hospital it was not present until a few hours before death. The blueness may be quite general, or may appear only in the lips, nose, eyes, fingers, and toes. In addition to the physical signs detailed above, one expects to find evidence of dilatation of the right heart in most cases of congenital cardiac disease.

Peripheral temperature is lowered. Dyspnea and cough are common symptoms. Such children are usually puny of body and stunted in mind. The fingers are markedly clubbed. The child fails to develop generally. A blood count usually reveals a remarkably large number of erythrocytes (stasis). (See chapter on Physical Development.)

**Diagnosis.**—The diagnosis of the exact condition is impossible in some cases, yet when cyanosis, cardiac hypertrophy, and murmurs are present, one can safely say that there is congenital cardiac disease present. We have briefly outlined the auscultatory signs of importance in the diagnosis of the most common lesions.

**Prognosis.**—This is grave in the majority of instances. A patulous foramen ovale is not incompatible with life, and 16 per cent. of the cases of pulmonary stenosis reach the age of twenty years (Assum).

**Treatment.**—This is essentially hygienic. "If," as Jacobi says, "they be so unfortunate as to grow up, exercise should be avoided." Warm clothing and a mild, equable climate will serve to prevent the internal congestions and the bronchitis, which may serve as fatal disturbances to a damaged and overlaid circulatory apparatus. Salt baths and inunctions of the skin with oil or fat are measures that appear of some practical aid in conserving nutrition.

Tonics will be of use as indicated, and digitalis may prove of service in crises. Oxygen and blood letting (venesection) would also be indicated in the presence of extreme cyanosis.

## FUNCTIONAL DISTURBANCES OF THE HEART

During the developmental epochs the heart is especially liable to disturbances of rhythmic action, but infants, as a rule, escape. Older children are thus troubled, however, especially as puberty approaches and during and after that time. Exciting causes, such as confinement indoors, a sedentary or overlaborious life, the use of improper food, tea, coffee, or tobacco, readily affect the growing tissues of so delicately poised an organ; also the effect of poisons of various sorts, as in the acute infectious fevers.

These affections are practically neuroses, and are not accompanied by demonstrable changes in structure; hence, are thus designated functional.

Wilson classifies the symptoms in the following table:

- I. Motor disturbances:
  - 1. Derangements of rhythm.
    - (a) Arrhythmia; (b) tachycardia.
  - 2. Momentary syncope.
- II. Sensory disturbances:
  - (a) Heart consciousness; (b) distress; (c) pain.
- III. Motor and sensory disturbances combined:
  - Palpitation.

Obviously this is but a classification of symptoms and throws no light upon the existing conditions upon which these symptoms are dependent. Indeed, arrhythmia may be noticed in perfectly healthy children during sleep, and, as before mentioned, the cardiac rate may be affected by trivial circumstances. On the other hand, some of these symptoms may be observed in organic heart or brain disease. We would recall attention to our brief summary of the physiology of the heart. It has been found experimentally that the rate, the force, and the rhythm may be affected by position, exercise, the condition of the cardiac muscle, the states of activity of the intrinsic and extrinsic nervous centers, the blood pressure, the degree of ventricular distention, acids and alkalis in the blood, the temperature of the blood, the quality of the blood, the condition of the coronary circulation, local heat and cold, drugs, also reflexes from various localities (intestinal, skin, genito-urinary system, etc.).

**Etiology.**—Anemia will be found as the most frequent cause of palpitation, etc.; tea- and coffee-drinking is far from an uncommon factor of disturbed cardiac rate and force; exophthalmic goiter is occasionally observed in childhood; so various disorders of metabolism with circulatory toxins are competent causes. Eye-strain, dyspepsia, gastro-intestinal fermentation, and nasopharyngeal growths will serve as examples of reflex causes.

Paroxysmal tachycardia, so thoroughly studied by Nothnagel, we have not observed. It must be remembered that many of the reported cases have displayed organic lesions of the heart or nervous mechanism in addition, and one should look carefully for such lesions before regarding such individuals as subjects of neuroses only.

Sansom reports 100 cases of influenza, with tachycardia in 37 cases, irregular heart in 25, and bradycardia in 5 cases. Our experience amply confirms the influence of the influenza poison upon the circulatory apparatus. Increase or slowing of the rate and abnormalities of rhythm frequently persist for a long time as resultants of this infectious disease. The irregularities in cardiac action so frequently seen

accompanying chorea, and which often pass away, leaving apparently no lesion, are in all probability instances of slight endocardial damage. These may not recur, but represent too often the initial lesion, becoming subsequently serious and disabling.

**Diagnosis.**—Our physiologic knowledge of cardiac structure, action, and normal variations should lead us to investigate etiologically, and not rest content with the diagnosis of a symptom. Our observations at the Polyclinic Hospital would lead us to believe that Da Costa's classification applies equally to children, and that many cases may be grouped under the heads of *muscular weakness* (*myasthenia cordis*) or *nervous weakness* of the heart. Of course, these conditions may be accompanied by general asthenic states. One could scarcely leave the subject of functional disorders of the heart without a reference to the so-called *functional* or *accidental murmurs*. As a rule, these are not heard in infancy, though the question is a moot one. These murmurs in childhood, particularly in older children, are of fairly common occurrence. Generally they are systolic in time. They are most frequently heard in the neighborhood of the pulmonic cartilage, and probably next at the apex. They are usually soft in quality, but may be quite harsh. Transmission does not pursue the laws followed in valvular disease, but exceptionally these murmurs are heard over considerable areas. Enlargement of the cardiac area is never present to any marked degree.

We can not afford the space to dwell upon the various theories advanced to explain these interesting murmurs; suffice it to say that no one theory satisfactorily accounts for all of these sounds.

**Prognosis.**—This will depend upon the gravity of the underlying conditions. As a rule, prognosis is decidedly favorable when organic cause is excluded.

**Treatment** must also depend upon the cause or causes of the symptom. In most cases it is largely hygienic. Attention to diet is always called for, as reflexes arising through the pneumogastric nerve—for instance, those from a dilated stomach—may mechanically influence the heart. If syncopal seizures are noticed, the child must be kept in bed, and there treated by massage, proper bathing, and a concentrated nutritious diet. Iron and arsenic used judiciously are of value in anemia. When the first sound of the heart is weak, or when tachycardia is accompanied by syncope, strychnin in small divided doses is our best remedy.

Hygienic measures are of most practical utility, including attention to the skin, lungs, bowels, and digestion, especially intestinal digestion

and absorption, and careful regulation of the bowels, avoidance of autointoxication from defective elimination.

The Bad Nauheim (so-called Schott) method of systematic bathing and of exercise against gentle resistance would seem to find its widest field in such cases. Even where the heart has been crippled by organic disease, however, good results have been produced. We would certainly regard the severer exercise of mountain-climbing as hazardous in the extreme in organic cases, and only to be approached by easy gradations, to be carefully supervised in any case. Gentle, largely passive, but always carefully regulated exercises will, if unaccompanied by excitement, prove of the utmost value.

### DISEASES OF THE PERICARDIUM AND ORGANIC CARDIAC DISEASES

**Pathology.**—It is customary in works on pediatrics to rest content with the statement that the pathologic changes do not differ from those observed in adults, but as the early periods of life so often mark the inception of these dangerous morbid processes, a concise review of the morbid anatomy will not be amiss.

Sturges is inclined to speak of **carditis**, believing that in severe cases the morbid changes are not confined to the endocardium or pericardium, but that these structures share with the heart muscle in a common pathologic process. As Crandall remarks, however, "these cases (of Sturges') came to autopsy," and we cannot feel certain that such diffuse lesions exist in most cases. Certainly disease of one or the other structure usually predominates, just as the croupous pneumonia will far outweigh the small amount of pleuritis that accompanies it.

**Pericarditis.**—Pericarditis is an inflammation of the serous covering of the heart and of its reflection on the inner surface of the pericardial sac. We recognize plastic pericarditis, pericarditis with serous or purulent effusions, and adherent pericarditis. The changes involved may be only different stages of one morbid process. On the other hand, plastic pericarditis may subside without other changes. Effusion may come on insidiously. Purulent pericarditis probably partakes of a purulent nature from the very onset. Adhesions may result in essentially chronic cases, in which there has been no suspicion of the process.

In plastic pericarditis the internal or external surfaces of the membrane may be affected, and the changes local or general. In the

mildest cases the membrane is red, sticky, and devoid of luster, the latter due to a light coating of fibrin. The fibrin may be present in such large amounts that the "hairy heart" (*cor villosum*) is produced. Some serous exudate is always found between the meshes of the fibrin. Many of these cases are tuberculous, the fibrin covering the small tubercles (Osler). If resolution occurs in this plastic stage, white spots are seen occasionally to dot the serous surface ("milk-spots"). The exudate may be simply serous, containing flocculi of fibrin and endothelial cells. This is the common form of effusion in rheumatic pericarditis in older children and in adults. Again, the fluid may be purulent, either because of extension from the contiguous mediastinal glands or pleura,



FIG. 41.—Chronic adhesive pericarditis.

as a manifestation of a pyemic process, or as a primary occurrence (local tuberculosis). When the fluid is bloody, the probabilities are that the affection is tuberculous. It may also be malignant. It will readily be seen that the rôle of the micro-organism in pericarditis is a most important one. (See Endocarditis.)

Adhesions may form between the two surfaces of the membrane (local or general), or extrapericardial adhesions may be present (adhesive mediastinitis, etc.).

**Myocarditis**, an inflammation of the muscular tissue of the heart, results whenever pericarditis is well marked. For a depth of two to three millimeters the muscle may appear quite pale (Osler). Affections of the cardiac muscle in childhood have not been well studied. One need scarcely more than allude here to the common forms of dilatation

and hypertrophy, whether these lesions occur independently or are accompaniments of valvular, pericardial, kidney, or pulmonary disease.

In pneumonia and diphtheria and scarlet fever cloudy swelling may be found, and in typhoid fever a true myocarditis may be present. Tubercle and syphilis rank as uncommon affections.

Sarcomata of the heart are exceedingly rare.

**Endocardial changes** may also accompany the aforementioned process, or occur without either. Micro-organisms may be found in lesions of the lining membrane, and yearly their number is increasing. We quote the following list from Wilson, though it is by no means exhaustive: Streptococci, staphylococci (aureus, albus, cereus albus, flavus, non-pyogenes), the bacillus typhi abdominalis, bacillus tuberculosis, bacillus diphtheriæ, bacillus pyogenes foetidus, bacillus of anthrax, the micrococcus lanceolatus, the gonococcus of Neisser, the diplococcus pneumoniae. Probably we may add the diplococcus rheumatica. In many of the cases of ulcerative endocarditis the gonococcus has been isolated from the valvular granulations; though it will be remembered that ulcerative endocarditis is rare in early life, and that though gonorrheal vulvo-vaginitis is all too common, it is rarely accompanied by endocarditis in little girls. We regard the list as very important. Inflammation attacks most frequently the valvular endocardium; in intra-uterine life the tricuspid, and in postnatal existence the mitral, leaflets being the preferred sites of disease.

The changes resulting are spoken of as verrucose (warty), ulcerative, or sclerotic; but, as in pericarditis, we can draw no sharp pathologic or clinical lines.

The changes occur two to three millimeters from the free margins of the leaflets (Osler)—*i.e.*, at the lines of maximum contact (Sibson). In the verrucose (warty) form small bead-like bodies or larger granulations are observed. These represent newly formed vascular tissue infiltrated with endothelial cells and capped by cellular debris and fibrin. Micrococci are present in ulcerative forms (Eberth and Klebs), but are known to bear no constant relation to the simple forms. These changes commonly pass into a sclerotic phase, with the tendency to contraction noticed in scar tissue generally. Should the resulting deformity narrow the valve orifice and offer resistance to the normal flow of blood, we speak of stenosis; should the valve leaflet fail to meet its fellows in closure, back flow or regurgitation would occur. Sclerosed valves are likely to be the seats of recurrent inflammations.

More rarely, indeed very rarely in childhood, the warty granulation undergoes rapid disintegration, the necrosed tissue is swept away, and

ulcerative or malignant endocarditis manifests itself. Such an ulcer may completely perforate a valve. Emboli are sometimes carried along into the circulation in the various forms of endocarditis, and the resulting phenomena observed in distant parts will vary according to the structures involved and the septic or non-septic characters of the débris.

## DISEASES OF THE PERICARDIUM

### PERICARDITIS

Inflammation of the serous envelop of the heart may occur, with or without muscular or endocardial involvement. It is properly considered a disease of the heart. Large effusions are more likely to occur in children than in adults.

**Etiology.**—Pericarditis is rarely primary, though it is true that the pericardium may be wounded from without, and that foreign bodies may ulcerate from the esophagus into the sac.

Rheumatism is the most frequent cause of secondary pericarditis; but the rheumatic affection may be so slight as to escape attention, the effects of the poison may be expended upon the pericardium instead of on the joints. Septic infection of the umbilicus ranks as a prominent cause in early infancy. Tuberculous forms of pericarditis are much more common than is usually supposed. Scarlet fever, typhoid fever, diphtheria, and measles are occasionally essential factors, but cases following influenza are reported, but it is difficult to exclude antecedent lesions. Pericarditis may be met accompanying or following many grave affections.<sup>1</sup> Extension of inflammation from contiguous organs occurs in a number of cases (pleuritis, mediastinal abscesses, pleuropneumonia).

Pericarditis may occur at any age, and males are more frequently attacked than females.

The bacteriologic findings in pericarditis are pneumococci the bacterium coli, streptococci, staphylococcus, and rarely the bacillus pyocyaneus. It is common to find it accompanied by endocarditis.

**Symptoms.**—The forms of pericarditis are acute and chronic; these are again divided according to the nature of the inflammation into simple, fibrinous, or plastic inflammation and inflammation with effusion. The lesions are further described as dry pericarditis, when the inflammation results in the output of little or no fluid, or if it is absorbed, leaving behind fibrinous bands; also purulent pericarditis,

<sup>1</sup>Baginsky met with pericarditis purulentia in phlegmonous erysipelas, grave forms of angina, caries of the ribs, fibrinous pneumonia, bronchopneumonia, gastroenteritis, furunculosis, phlegmon of the throat, and empyema.

when the fluid is infected by pus from any source. (See Pathology.) Very frequently in *plastic pericarditis* the condition is not suspected during life. Pain referred to the precordia or to the xiphoid cartilage may or may not be present. There may be left pleurosthotonus—a bending of the body to one side. The pulse is usually full and rapid. Slight fever is common, and a hacking cough is often present. If effusion should form in any considerable amount, sharp or stabbing pain be complained of, or the patient simply experiences a sense of discomfort in the precordial region. Here tenderness at the lower sternum may be coupled with the pain. Dyspnea is common, and should always demand a careful examination of the heart and lungs. The face may be dusky and the expression anxious. The patient is restless; the pulse is rapid, small, is sometimes irregular, and the pulsus paradoxus (failure or weakening of the pulse during inspiration) may be observed.

Pressure on the recurrent laryngeal nerve, when present, causes aphonia, or the left lung may become physiologically hampered from the pressure exerted.

Syncope, hiccup, insomnia, and low delirium are present in the severer cases; or marked cerebral symptoms may manifest themselves in the hyperpyrexia of rheumatic sufferers.

Effusions may come on insidiously, and with practically no symptoms; if abundant, cyanosis and orthopnea may be graphic features.

In adherent pericardium the symptoms are uncertain and indefinite. The affection may not be suspected until a careful physical examination demonstrates great cardiac enlargement.

**Physical Signs.**—*Inspection* reveals an overacting heart in the plastic form of pericarditis. When effusion takes place, the precordia bulges, the intercostal spaces become prominent, and edema of the thoracic wall may be observed (especially when pus is present). The displaced viscera of the abdomen may produce a prominence in the epigastrium. Systolic retraction at the apex, diffusion of the apex-beat, and Freidreich's sign (diastolic collapse of the cervical veins) are of diagnostic importance in adherent pericardium. Much more important than retraction anteriorly is the posterior retraction described by Broadbent and known as "*Broadbent's sign*." (Below the inferior angle of the left scapula.)

**Palpation** may reveal a distinct friction fremitus in simple pericarditis, most marked over the right ventricle, about the fourth interspace, and best felt as the patient leans forward. As effusion progresses the cardiac impulse becomes weakened and finally lost. The apex-beat

seems displaced upward and outward, though this is probably apparent and not real. (Rotch contends that some other portion of the heart comes in contact with the chest wall. He has probably proved this contention.) At the same time the pulse may be quite forcible. Fluctuation can rarely, if ever, be detected. When adhesions form, systolic retraction can in some instances be felt at the apex, and a diastolic rebound may follow it.

**Percussion.**—In simple pericarditis or commencing effusion we can expect to find nothing but tenderness on percussion. As the fluid exudate increases the precordial area of dullness becomes much enlarged, assuming a pear-shaped type with the base directed downward and its apex toward the manubrium, the reverse of normal. Rotch lays great stress upon the enlargement to the right of the sternum, considering this diagnostic. Sansom finds a small posterior area of dullness important.

**Auscultation.**—Most pathognomonic of plastic pericarditis is the friction sound. It is a to-and-fro or double sound, corresponding to systole and diastole, but outlasting these periods. Its superficial character is distinctive. The sound usually exhibits a rubbing or grating quality, but may simulate the creaking of new leather (*bruit de coif neuf*). It is usually present over the right ventricular area, but may be noticed at various sites. It may simulate certain valvular murmurs, but does not follow the laws of transmission. When fluid appears in quantity this sound usually disappears, or is heard only in certain limited areas. With the absorption of fluid it may reappear. In large effusions auscultation over the left lung may reveal feeble or tubular breathing.

In chronic adhesive pericarditis a loud systolic murmur may lead to an error of diagnosis. On the other hand, murmurs are sometimes absent. The fetal rhythm is heard when marked dilatation is present.

The pleuropericardial friction is a duplex phenomenon in which altered sounds accompany the respiratory as well as the cardiac movements.

**Diagnosis.**—In cases of frank articular rheumatism, where the heart is examined daily, the onset of pericarditis should not be difficult to recognize. But rheumatism is so frequently insidious in the child that this cause may not be traceable. Pericarditis complicates other affections, and as the pericardial inflammation may give rise to very little disturbance, the affection is often overlooked. When effusion is excessive, or when extensive adhesions cripple the heart, the diagnosis may be most obscure.

Cardiac dilatation or hypertrophy or enlargement of the heart from any cause may be most difficult to distinguish from pericardial effusion. The Röntgen rays will prove of the greatest value in the differentiation of these two conditions. A weak or absent apex-beat, coupled with a strong pulse, should strongly suggest effusion. Again, the shape of the dull area is of great importance, being increased in all directions—in the majority of instances in the form of a pear.

In dilatation the impulse is visible and wave-like; it is scarcely visible in pericardial effusion.

The double murmur of aortic valve disease may simulate a friction sound.

The systolic murmur in adhesive pericarditis may be difficult to distinguish from the mitral systolic murmur of regurgitation. A study of transmission and quality of sounds over various portions of the chest will here enable us to differentiate. Lastly, certain cases of massive effusion may be most difficult to diagnose, as they simulate, even to the tubular breathing, left-sided pleural effusion.

When purulent pericarditis is suspected, diagnostic puncture should be performed. A temperature-range peculiar to a purulent process and a study of the blood will assist the observer.

**Prognosis.**—This depends largely upon the etiologic factors and upon the amount of effusion and whether it is serous or purulent. In rheumatic pericarditis the immediate outlook is usually good, though these cases with large effusions may die suddenly or the heart be subsequently crippled by adhesive bands. In septic or purulent pericarditis the prognosis is most gloomy. Tuberculous pericarditis, though slower in its course, also terminates fatally.

**Treatment.**—In acute pericarditis the child must be kept at absolute rest in bed, and free from all psychic or other disturbances. This condition of physical and mental quiet must be maintained for weeks or months. Locally, dry cold should be applied, as this suffices to lessen the cardiac rate and vascular pressure. Heat (dry or moist) may supplement this with much comfort and advantage at certain times. The diet should be simple and concentrated, peptonized foods being demanded where there is gastric disturbance; all gaseous distention must be promptly relieved. The systemic treatment will depend somewhat upon the accompanying and causative affection. In rheumatism alkalies combined with the salicylates should be given, unless there is great depression. In septic conditions active stimulation is demanded in spite of the pericardial complication. Morphin is the stand-by to relieve pain and great restlessness, though phenacetin in small doses

may be useful in mild cases, and chloralamid or sulphonal will control restlessness when opium is not demanded. When the inflammation subsides and the effusion appears upon the increase, small blisters applied over the precordia at intervals of seventy-two hours are occasionally very useful. Calomel, alone or combined with Dover's powder, is a valuable agent here. A dose or two of a saline laxative is often most useful in robust children or sthenic patients. Potassium iodid is recommended in this affection. Caffein, spartein, or diuretin will find use in certain cases. Digitalis, strophanthus, and convallaria are to be used only when there is marked cardiac weakness. It is well to begin with aromatic spirits of ammonia before using the more powerful cardiac tonics.

In massive effusion paracentesis should be performed, either in the fifth interspace, slightly to the left of the sternum, or, as Rotch suggests, to the right of sternum. In serous effusion aspiration will prove sufficient, but where pus is suspected, a surgeon should always be called, and in this grave affection we cannot regard any hopeful operative measure as too radical. Epileptiform seizures or choreiform movements may appear during the operation of paracentesis.

#### OTHER AFFECTIONS OF THE PERICARDIUM

**Hydropericardium**—a collection of serum within the pericardium—in the child is quite rare; it is most likely to occur in kidney disease, and more rarely (unaccompanied by other dropsical symptoms) it is observed after scarlet fever.

**Hemopericardium** (blood within the pericardium).—The productive factors of this condition in adult life do not obtain in the child, though, as already mentioned, tubercle may be accompanied by sanguineous pericardial exudate.

**Pneumopericardium**—air within the pericardium—may be produced as in the adult, and differs in no way from the condition observed in adult life.

#### AFFECTIONS OF THE MYOCARDIUM

**Etiology.**—Hypertrophy and dilatation usually occur conjointly, and may result from excessive cardiac activity *per se* (as in exophthalmic goiter); mechanically, from extracardiac adhesions or as compensatory efforts in valvular disease. Nephritis is a cause of left ventricular *hypertrophy*.

**The symptoms** of myocarditis can scarcely be separated from those of accompanying inflammatory conditions. *Excessive dyspnea, cyanosis, and palpitation* would render that diagnosis probable. Dilatation and hypertrophy may or may not be accompanied by symptoms. When the former condition far exceeds the latter (*dilatative hypertrophy*) circulatory phenomena arise, which will be recognized as evidences of failing compensation.

In syphilis or tubercle we should expect to find other features or signs characteristic of one or the other disease.

### MYOCARDITIS

Myocarditis, if we are to form an opinion from the various textbooks, would seem to be one of the rare diseases of childhood. Judging from the observations made at autopsies, where, indeed, most of our knowledge of the pathology and theory of the disease is obtained, it is much more frequent than is generally supposed, and, while an important factor in the symptomatology of infectious diseases, it is not so fatal as the literature on pediatrics would lead us to infer.

Myocarditis is an acute or chronic inflammation of the muscular structure of the heart. The chronic form is always found in adults and associated with sclerosis, and therefore will not be discussed here.

**Etiology.**—Myocarditis is either primary, when due to diathetic dyscrasias—such as congenital syphilis, tuberculosis, etc.; or secondary, when due to endocarditis, pericarditis, toxins from the infectious fevers, or poisons, like lead, arsenic, or phosphorus. It may be of traumatic origin. Boys are much more liable to be affected than girls.

**Pathology.**—Macroscopically the heart muscles are pale, soft, and friable. Microscopically, changes are found in the parenchyma: cloudy swelling, fatty infiltration and fatty degeneration, with an invasion of the connective tissue by leukocytes. The whole structure of the heart is not always affected, and some portions of the cardiac tissues may be quite normal, while others have become degenerated. The myocardium is peculiarly susceptible to the toxins of infectious fevers; the fever and the disease itself may seem to play but a subordinate part in fatal cases. This is particularly true in diphtheria, scarlet fever, typhoid fever and pneumonia, where the severity of the toxemia may bear no or only a small relation to the apparent mildness of the disease. Autopsies in diphtheria, pneumonia, scarlatina, and typhoid fever constantly show sufficient myocardial changes to have caused death. When cloudy swelling and fatty infiltration have occurred, complete recovery may follow, but when further degenera-

tions have invaded the tissues, permanent injury results. The former conditions are constantly found in the infectious fevers.

**Symptoms.**—The symptoms of myocarditis are apt to be lost sight of in the care of the primary disease, if indeed any are present. Some of the most serious cases of myocarditis have been evidenced only by the sudden death of the patient. When, however, faintness, cyanosis, vomiting, dyspnea, and precordial pain or distress, together with a weak, rapid, irregular heart action, with a feeble impulse and weak first sound, are present, it is certainly significant of myocarditis, and should be so considered, especially when associated with or following upon the infectious fevers. In these cases again we find the ratio of the pulse and respiration distorted. Dilatation and hypertrophy may or may not be accompanied by symptoms. When evidences of failing compensation, with its attending circulatory phenomena, arise, eccentric hypertrophy can be recognized.

**Diagnosis.**—The diagnosis of myocarditis is most often made first at the autopsy. A positive diagnosis during life is generally impossible, but should always be suspected in the infectious fevers, when the cardiac symptoms, before mentioned, are present, especially so when pericarditis and endocarditis can be excluded.

**Treatment.**—Absolute rest should be insisted on, especially following severe attacks of the infectious fevers, when myocarditis is almost always a coexistent condition. The patient should be kept in a recumbent position for several weeks, and prevented from making any sudden exertion, which might prove fatal. Cardiac stimulants like ammonia, alcohol, caffein, and strychnin are used symptomatically, small doses of strychnin being depended upon for continuous use. The intermittent use of inhalation of oxygen, if employed early enough, is capable of saving lives. Digitalis should be used with caution and only when evidences of muscular failure are plain. When symptoms of heart failure appear, morphin hypodermically may be administered with gratifying results. Syphilitic and tubercular myocarditis demand appropriate treatment.

**Sequelæ.**—Those cases where cloudy swelling or fatty infiltration only has taken place may go on to a complete recovery, when the heart tissue resumes its normal condition. In extreme cases, where the toxemia has been intense, heart failure may terminate the case; but when fatty degeneration has occurred, where the proper nutrition of the heart muscle has been interfered with, there is a permanent structural change. This results in a hypertrophy and dilatation, and may even proceed to an aneurysm or rupture of the heart.

**Acute suppurative myocarditis**, or abscess of the heart, is a rare condition in adults or children. When seen, it is due to a phlebitic or pyemic origin.

**Cardiac aneurysm**, while not always, is generally due to a true myocarditis, and its favorite point of election is the left ventricle. It may reach a considerable size. Embolism into the coronary artery may lead to this condition by causing a necrosis of the tissues. It is extremely rare in children.

## DISEASES OF THE ENDOCARDIUM

### ENDOCARDITIS

#### Synonym.—VALVULITIS

**Etiology.**—Rheumatism is by far the most frequent cause of endocardial inflammation. Chorea is frequently accompanied by endocardial change, but here again the underlying cause is probably rheumatism in the majority of cases. Septic conditions give rise to endocarditis. Pneumonia and pleuritis are frequent causes. Scarlet fever is too often complicated by endocarditis; and diphtheria, measles, typhoid fever, and variola rank as occasional causes. Tuberculous endocarditis occurs. Endocardial changes may accompany acute or chronic nephritis. Tonsillitis is another disease that may give rise to endocarditis. Lastly, endocarditis recurrens, rather a frequent affection, is worthy of attention. According to Crandall, girls suffer from rheumatism and consequent valvulitis much more frequently than boys. Endocarditis *may* occur *in utero*, but is rare under five years.

**Clinical History.**—Acute endocarditis may be cured—"really cured" (Jacobi)—in the child, but too often the acute inflammation ends in the sclerotic changes previously described, and we have the crippled leaflets of chronic valvular disease. This may never cause any inconvenience in the subject affected, for the muscular walls of the chamber or chambers, which must stand the brunt of strain, undergo a true hypertrophy.

Thus, in partial obstruction at the mitral valve we should expect left auricular hypertrophy. In regurgitation at the same orifice the left ventricular wall would share in the enlargement, for it must now exhibit force enough to send an increased amount of blood in two directions. The same ventricular wall would hypertrophy in the common combined lesion at the aortic valve. Where such hypertrophied muscle adequately performs its increased task, *compensation* has

been effected. Now, if the heart muscle of the left side fails to compensate fully, the strain comes upon the pulmonary system of vessels, and certain lung symptoms arise. The next tissue affected would be the right heart; and, lastly, the strain would manifest itself in back pressure upon the venous system. Fortunately, compensation is very readily effected in the child, and so even in grave endocarditis children may thrive surprisingly. We must not forget, however, that *recurrent* inflammation is common, and that during such recurrence some acute disease or physical strain may serve as the immediate determining factor of *failing compensation*.

Practically, then, we have three clinical stages of endocarditis: (1) Acute inflammation, which may terminate in recovery, death, or chronic valvulitis; (2) compensation (in chronic valvulitis); (3) failing or lost compensation.

**Symptoms.**—The symptoms of acute endocarditis may be obscure or wholly wanting, so that a diagnosis is not made until permanent damage is done. When it arises in the course of an acute disease, as articular rheumatism, the temperature rises slightly ( $1^{\circ}$  to  $2^{\circ}$  F.), the pulse-rate is increased, and the child is restless, with anxious expression. Pain and palpitation are rarely experienced. Cyanosis may appear, especially if the myocardium become involved (*vide supra*). The occurrence of fibrous nodules around joints is suggestive, though not pathognomonic. Advanced symptoms are seldom seen in the first attack. However, there are certain grave cases of rheumatic endocarditis which are attended by high fever, marked constitutional symptoms, and hemorrhages, thus making one think of ulcerative endocarditis (Litten). Lastly, ulcerative endocarditis itself is usually accompanied by marked cardiac disturbance, by a typhoid state, by purpura and hemorrhage from mucous membranes, and by the presence of embolic abscesses in various parts of the body. Fortunately, this fatal disease is rare in childhood.

If, to meet the damage done, compensation is fully established, chronic valvular disease may be devoid of symptoms; but if compensation should be imperfect, some of the following symptoms are liable to be present: *Dyspnea* and *palpitation* are most common in aortic disease; *pain* is rarely present, except in mitral stenosis; *typical ascending edema* is almost never seen in children, although *pulmonary congestion* is common enough; *epistaxis* is far from rare; *subacute bronchitis* and *persistent cough* are frequent accompaniments of chronic valvular disease. *Cyanosis* and *venous stasis* especially accompany mitral regurgitation.

**Physical Signs.**—Acute endocarditis is usually diagnosed by the physical signs. Inspection shows a rapid and diffuse cardiac beat. Palpation confirms this observation, and may reveal a strong, somewhat jerky, or irregular pulse (*pulsus celer*). Percussion is at first negative, unless some preceding disease of the heart has induced a change in its volume. The signs of dilatation appear sometimes with astounding rapidity. On auscultation we may hear, usually at the cardiac apex, a large blowing systolic murmur or souffle. When we remember that the localized damage may be exceedingly insignificant, we are not surprised that physical signs occasionally fail us. Osler styles these signs “notoriously uncertain.” In severe cases of ulcerative endocarditis, however, the physical signs are likely to be accentuated.

In chronic valvular disease a careful and judicial study of physical signs is of the greatest diagnostic and prognostic importance. We have outlined certain limitations in the study of these conditions in an early chapter; we will speak of others now, but these need not discourage us from carefully studying every case. The research is a more difficult one in children than it is in adults, but it is still a fruitful and imperative quest. We have no more patience with the diagnostic Nihilist than with the therapeutic brother of the same negative type. The tools at hand are simple and effective in each instance; one has simply to acquire a practical knowledge of their judicious use.

We shall exclude *pulmonary stenosis* from our consideration here, as it is almost invariably a congenital affection, and has been described as such. There remain for our study mitral regurgitation, mitral stenosis, aortic regurgitation or double aortic disease, and tricuspid regurgitation.

**Mitral Regurgitation or Insufficiency.**—The leaflets of this valve suffer most frequently from endocardial inflammation, and incompetency (producing regurgitation) is the most common result.

*Inspection* frequently reveals a bulging of the precordia. The apex-beat appears diffuse and may be displaced downward and to the left. The cervical veins may be distended and occasionally they exhibit pulsation. *Palpation* furnishes us with our best means of locating the apex-beat, and this is usually dislocated downward and to the left (Sansom). Pulsation may be quite general and diffused, felt over the whole left ventricular area, and usually over the right ventricular region.

One should mentally note whether such pulsations are weak and wavy or forceful. A systolic thrill is sometimes present, as in any valvular affection, but thrill is much more common in mitral stenosis.

In no valvular affection does *percussion* reveal so broad an area of dullness as in well-marked mitral regurgitation. The dullness extends to the left, beyond the nipple-line, possibly to the axilla, and to the right as far as the right sternal border or beyond it. But percussion should not be performed to ascertain the *size* of the heart alone; percussion reveals most when it informs us of the sizes of the various chambers of the heart (Sansom). Thus we are led inductively to the study of *auscultation*, which, though our most nearly perfect method of exploration, is often but confirmatory of what the other procedures have already made

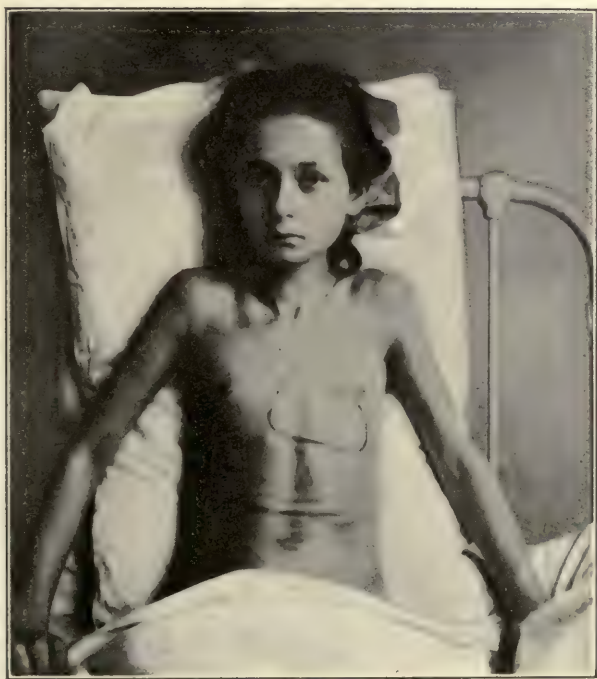


FIG. 42.—Double mitral-disease with failing compensation in an eleven-year-old girl. The patient has had rheumatism several times, and has been known to have cardiac disease for two years. Note the slender arms.—(*The Samaritan Hospital.*)

clear. Before attempting to study murmurs, we should carefully estimate the intensities of the first and second sounds. Now, murmurs are not always present in valvular disease, but, on the other hand, most murmurs in childhood are organic. One must study the point of *greatest intensity* very carefully, for the child's chest is so small that sounds are often widely diffused. The murmur of mitral regurgitation is almost invariably systolic in time, and the sound con-

tinues throughout the systole. It is heard best at the apex, though occasionally the base or midsternum may present the loudest sound. It is transmitted to the axilla and to the left scapular region. The quality may vary from a soft cooing murmur up to a murmur of a harsh rasping or sawing character. We have plainly heard this murmur with the examining ear an inch from the chest. Double mitral disease is rather frequently observed.

**Mitral Stenosis or Obstruction.**—This affection is much less common than the foregoing lesion, though a considerable degree of stenosis may accompany regurgitation (Fig. 42). Mitral stenosis arises in insidious forms of rheumatism (Sansom; Crandall).

*Inspection.*—Pulsation may be observed over the upper chest upon the left side. Osler states that this is due to right ventricular hypertrophy, no matter how far upward and to the left it may extend. The apex appears normally situated or is removed but a short distance from its usual site.

*Palpation* reveals pulsation over the left auricular area and over the right heart. An apical presystolic thrill is quite common. This may be present at certain times and not at others. It is usually accentuated by an upright or bending forward position. In well-marked instances this sign is practically pathognomonic. *Careful percussion* reveals dullness, extending to the right and in an upward direction to the left of the sternum. Hypertrophy is seldom so marked as in double mitral disease or mitral regurgitation. Tyson states that theoretically one would expect the left ventricle to really diminish in size. The murmur of mitral stenosis is absolutely characteristic. It is of short duration, occurs during the presystolic or later diastolic period, becomes rapidly intensified, and ceases with the systolic impulse. It corresponds in time to the described thrill. It is best heard at the apex or in the fourth interspace above. Ordinarily it is not transmitted to any degree, though Griffith has observed cases where this murmur was heard in the axillary region and back. Our studies have confirmed the statement that the area of the murmur is singularly restricted. Reduplication of the second sound and gallop or fetal rhythm are rather common in mitral stenosis. Accentuation of the second pulmonic sound is invariably present. The murmur described above is singularly inconstant: it may be present at one time and not at another, so that too much prognostic expectation should not be founded upon its disappearance.

**Disease of the Aortic Leaflets.**—As alcoholism, excessive and continuous muscular strain, and acquired syphilis are rarities in childhood, the etiology of aortic disease must differ essentially from that of

the adult. Excluding congenital cases, the disease is almost invariably due to rheumatism. As a single lesion, it is very rare in childhood.

In **aortic regurgitation** or **insufficiency** inspection reveals the apex dislocated to the left and to a low point in the thorax. The apex has appeared as low as the eighth or ninth interspace in this disease (*cor bovinum*). The precordia bulges markedly in the young child, especially over the left ventricular area. The arteries in the neck are seen to throb violently, and the brachials often plainly exhibit the phenomenon. Even the radials appear to fill and empty most rapidly. The characteristic quick pulse of aortic regurgitation is known as the Corrigan or water-hammer pulse. In the larger blood-vessels the quick systolic impulse and equally sudden collapse may be even more apparent ("steam-boat" pulse).

The capillary pulse is another phenomenon of aortic regurgitation. The ophthalmoscope reveals pulsation of the retinal arteries. Palpation confirms the suspicion of a downward dislocation of the apex-beat, and probably a throbbing sensation can be perceived over all the precordia. A thrill at the aortic cartilage or in its vicinity may be felt. Percussion reveals a much enlarged precordial dullness, both absolute and relative, and the increase in the longitudinal diameter predominates over the lateral increase. (See Mitral Regurgitation.) The left ventricle may be alone affected, or all of the cavities may suffer alike from dilatation and hypertrophy (*cor bovinum*).

On auscultation over the affected heart one hears a diastolic murmur at the aortic cartilage (second right interspace). The sound is usually a soft bruit, long drawn out. Frequently it completely replaces the valvular click of the sound, though this is not always the case. It is transmitted down the sternum or toward the apex. A sharper murmur, systolic in time, may precede the regurgitant murmur, and is probably produced by roughening of the leaflets; for in these cases the valve orifice is so dilated that stenosis is out of the question.

**Double Aortic Disease.**—While regurgitation is not always accompanied by stenosis, yet marked stenosis is probably accompanied by leakage in every instance. Thus we deem it proper to speak of double or combined aortic disease. Of course, one or the other of the conditions may preponderate. Marked stenosis is exceedingly rare in childhood except as a congenital lesion. It is scarcely necessary to mention that we may have a combination of aortic, mitral, and tricuspid disease.

*Inspection.*—The apex-beat may be somewhat displaced, but not nearly so much as in the regurgitant lesion.

*Palpation.*—A somewhat forceful and disseminated beat is

usually felt. A *thrill*, systolic in time, is decidedly characteristic. It is very marked over the base of the heart or at the aortic cartilage.

*Percussion* shows slight enlargement of the left ventricle (excentric hypertrophy), mayhap also involvement of the right heart.

*Auscultation*.—Unless the systolic murmur is exceedingly shrill and harsh; unless it is heard best at the second right interspace and is transmitted into the large arteries of the neck and axilla, we cannot be certain of aortic stenosis. The diastolic murmur is not always present. In a case recently seen at the Polyclinic Hospital the aortic sound of closure was perfect and there was no murmur in diastole. Sometimes a typical to-and-fro murmur is heard. (See Aortic Regurgitation and Pericarditis.)

**Tricuspid Disease.**—Regurgitation is the only lesion worth considering, as stenosis is nearly always congenital and the patients thus affected soon die. Regurgitation at this orifice is primarily due to involvement of the left heart or to pulmonary disease, the right side suffering secondarily. It may occur in chronic bronchitis or in congenital bronchiectasis, and is accompanied by systolic pulsation of the cervical veins and pulsation of the liver. Signs of right cardiac hypertrophy are demonstrated by percussion.

*Auscultation*.—The murmur is heard near the lower portion of the sternum, and is soft in character and systolic in time. It may be impossible to distinguish it from the murmur of mitral regurgitation.

*Diagnosis*.—If in every case of rheumatism and in cases of infectious disease an examination of the heart should be made daily, the diagnosis of acute endocarditis would be secured more frequently. In pericarditis the friction sound should serve to differentiate. Cases of ulcerative endocarditis are frequently mistaken for typhoid fever; the diagnosis is sometimes very difficult, and we should endeavor to exclude all diseases with similar symptoms. (In a case at the University Hospital miliary tuberculosis simulated ulcerative endocarditis.) In cases of chronic heart disease physical signs, carefully weighed, will serve us in most cases. We must not rest content, however, with the diagnosis of a lesion, but must also appreciate the ratio between hypertrophy and dilatation, and must study carefully the general condition of the patient.

*Prognosis*.—In acute endocarditis the prognosis is usually good. In the severe recurrent types we must judge of severity by the amount of myocarditis and the intensity of the symptoms that have been detailed. In ulcerative endocarditis the prognosis is absolutely gloomy.

In dealing with **chronic cardiac disease** the quasi-scientific man

blunders most miserably. Let him not assume that the presence of a heart murmur is necessarily of dark portent, nor that it should become a bugbear during the remainder of the patient's existence. Judge carefully of the amount of enlargement of the heart; try to estimate whether hypertrophy is present to a compensatory degree. Above all, do not forget that one is called upon to pronounce an opinion upon a patient and not upon a heart. Recurrent rheumatic attacks, poor hygienic surroundings, lowered nutrition, puberty, etc., are damaging factors. On the other hand, the maintenance of perfect nutrition and of perfect cardiac compensation may obtain for many years. The outlook, in the presence of good general conditions, is almost always bright for the child. It will be most unfavorable in aortic incompetency, especially if failing compensation be present, somewhat better in mitral stenosis, best in mitral regurgitation.

**Treatment.**—In acute endocarditis our first effort must be directed toward minimizing the amount of work done by the affected heart. Perfect rest to body and mind is, if anything, of more importance than in pericarditis. The child should be placed in bed and kept there, unless excessive fretfulness forbids, when the nurse's arms or a comfortable lounge may serve in good stead. The diet should be light, digestible, and given in small amounts and at rather frequent intervals. Milk will prove the best food. If it produces flatulency, predigestion or dilution or both should be employed.

Acute diseases of the heart, as a rule, are inflammatory; but not always. In many conditions where the cardiac disease is a frequently occurring complication, as in diphtheria, the danger is from a parenchymatous myocardial degeneration. This is true also in children during attacks of the exanthemata, in pertussis, and in pneumonia. Here the deaths are due primarily to the involvement of the heart muscles, which are sometimes fatally weakened by the toxins of these infections without giving any discoverable signs of carditis. In them, too, a specific softening and consequent destructibility or acute structural dilatation of the myocardium generally takes place, which may be only, or best, revealed by percussion. Evidences of a general failure in the circulation must not be overlooked. Overexertion after the continued fevers is a fertile source of cardiac damage of the same character as that following prolonged and severe strains upon the normal heart.

In inflammatory cardiac conditions there is great excitement of heart action; the muscle is in ceaseless activity, and during this period it is doing twice as much work as in health; hence every effort must impera-

tively be made to maintain absolute rest of mind and body in every form of carditis.

It is clear that at this stage no tonic medication is of use, but only quieting agents are indicated. If pain is present, clearly evidenced or to be elicited by pressure under the left costal arch, along with hurried breathing, rapid pulse, disturbed rhythm, with or without murmurs, the treatment should be external applications which will give comfort and quiet to the heart, such as hot poultices of linseed meal, to which may be added laudanum or belladonna. Local blood-letting by leeches applied to the sternal notch, thus causing a reflex nervesedation, produces much relief in older children. The ice-bag to the chest will prove a useful local measure, especially if pain is present. When the heart is acting well and good compensation has been established, as shown not only by the normal position of the apex-beat but also by the absence of signs of backward pressure in the lungs, liver, etc., drugs that would further stimulate the heart are obviously harmful. If the physical signs are not associated with any evidence of failure of the cardiac muscles, the effect of routine treatment by digitalis upon the heart would be to drive the myocardium to increased effort; the endeavor of the right ventricle to force the blood through the lungs more rapidly might possibly lead to hemorrhage from the pulmonary capillaries, and the left auricle as well as the right ventricle would probably further dilate.

Treatment should be directed to the maintenance of the balance in the circulation by judicious advice as to the manner of life to be led and the attention to the general health. The heart should be carefully examined at intervals, especially when any signs of downward progress, such as increased or increasing dilatation or engorgement of the liver and lungs, begin to make their appearance. If, however, these symptoms show themselves, now is the time of all others that drugs of the digitalis group are indicated and form a necessary factor in the treatment. As a preliminary to the use of digitalis a free purgative is very useful, and leeches to the precordia, or even venesection, may with advantage be resorted to when there is cyanosis. The effect of these remedies upon the heart and circulation should be carefully watched. When larger doses of digitalis are called for, it is well to restrict the patient to the recumbent position.

There is a most intimate vasomotor association between the nerves which contract the blood-vessels of the viscus and the cutaneous nerves of the corresponding skin area thereabouts (W. H. Thomson).

Systemically, in rheumatic cases we should use the salicylates alone

or combined with alkalies—strontium salicylate is perhaps the best. These should never be used long at a time—not over two or three days continuously—for it is quite generally recognized that endocarditis is an incident and not a complication in the course of rheumatism. Jacobi lays great stress upon the use of potassium iodid. Phenacetin is also a helpful drug, and, aside from its effect upon the fever and pain, is probably antirheumatic. The bromids are useful to control restlessness. In serious cases, attended by great pain and restlessness, opium is again the remedy *par excellence*. Alcohol and cardiac stimulants are contraindicated in simple cases. The bowels must be kept rather freely open by the use of mild laxatives or enemata.

If a surface pallor with a small, rapid, irregular pulse is noticed, it may be the result of a myocarditis and arterial degeneration due to the toxic effects of the exanthemata and diphtheria. Digitalis is then of no use, because it will not benefit the degenerated muscles, but rather requires a fairly normal muscle to work upon. Here strychnin is a far better agent, along with some alcohol. The hypodermic use of camphor is also of service; three to eight grains may be given in sterile oil to a child of five.

When the more protracted effects of an endocarditis are present, it is well to insist upon several weeks of absolute rest. There may be much relief thus afforded to the inflammatory vegetations which tend to form on the valves and flaps, by keeping the circulation as quiet as possible, otherwise there is danger of adhesions with contractions of the delicate endocardial structures.

In septic cases (ulcerative endocarditis) stimulants must be used with a free hand in spite of their local effects on the heart.

After a pericarditis it sometimes happens that the inflammation extends to adjacent structures, such as the pleura, ribs, or sternum, and adhesions result. Here mechanical measures are useful, such as strapping the left side with strips of rubber plaster, in order to limit the full force of the systole and assist in maintaining tranquillity. If there is pain also, heat or cold may be used, along with belladonna ointment externally.

It is in chronic heart disease that ardent therapy may carry havoc in its path. These cases should be recipients of hygienic care, as regards diet, bathing, exercise, and rest; and children should be guided into such life pursuits as will minimize the demands of physical efforts and strains. It will be well for every student to read Jacobi's sound advice in his most excellent book on "Therapeutics of Childhood." If compensation is adequate, the heart needs no drug to whip it on to

increased endeavor and consequent disturbance of balance. Some member of the family who will aid us in enforcing proper hygienic and other limitations must be informed of the cardiac condition. Lastly, each case must be judged on its own individual indications, and our efforts must be largely guided by results obtained. When the cardiac balance is disturbed, once more *rest* must be enjoined. Often this masterly inactivity alone will prove sufficient to reestablish the equilibrium so necessary to secure in such cases. When cyanosis, dropsy, pulmonary congestion, or other serious symptoms arise, digitalis is by far the best drug at our command. It may be used irrespective of lesion, though it probably does best in mitral regurgitation. The infusion is the best preparation, and may be given in doses of from twenty minims to one fluidram, or more, according to the age of the child. Strophanthus may be used where symptoms of digitalis poisoning arise or where digitalis cannot be given. Spartein, caffeine, convallaria, nitrites, etc., are indicated in certain cases, but they are all vastly inferior to digitalis. Digitalis may be given for weeks, or even months, until compensation is restored. Iron will prove of great value during and after convalescence. Strychnin in small tonic doses is especially useful when the myocardium is weakened. Cod-liver oil may also be of use.

There comes a time in subacute and chronic disease of the heart when carefully systematized exercises, such as breathing, posturing, and later passive and slightly active movements, are of great utility to improve the circulation and maintain vigor, both mental and physical.

## DISEASES OF THE BLOOD-VESSELS

### Arteriosclerosis

**Etiology and Pathology.**—It is not surprising that vascular disease should be uncommon in early life; for is it not common knowledge that “a man is as old as his blood-vessels. Nevertheless, cases of arteriosclerosis (even with atheromatous plates) in infancy and childhood have been reported by several authorities. Galli lays stress upon hereditary tendencies in these cases (*aortisme hereditaire*). Probably syphilis is more frequently responsible for arteriosclerosis than any other disease; indeed, one of us has come to view tortuous and hard temporal arteries in infancy as pointing strongly toward the existence of this constitutional vice. Morgan recognizes a rheumatic form of aortitis in early life. We have already spoken of the arterial changes that may take place in chronic paren-

chymatous nephritis. Many vessels may be affected, even those of the brain.

**Symptoms.**—With aortitis one may note retrosternal pain, dyspnea or even asthmatic attacks (De Gassicourt). The **diagnosis**, however, must usually be made upon the physical finds. Over the manubrium sterni may be heard a systolic murmur that is transmitted into the great vessels. This must be differentiated from the murmur in stenosis of the aortic valve. In the peripheral vessels the diagnosis is made as in later life.

The **prognosis** will depend largely upon the cause, syphilitic cases giving the best opportunities for successful therapy. The **treatment** is the same as in adult life, iodids being the most valuable drugs.

**Aneurisms of the Heart and Vessels.**—Both have been reported, though both are rare. This is not surprising when we find that arteriosclerosis is so rare. They have been observed in the fetus, however, and an aneurism of the ductus arteriosis in a new-born child (Riesman). They are most commonly observed between ten and fifteen years of age. Jacobi collected twenty-eight cases, but only twelve occurred under fourteen years of age. One of these was an aneurism of the abdominal aorta, the other were all aneurisms of the arch of the aorta. Another site of preference is the concavity of the aorta where the ductus Botalli is given off. Syphilis is believed to be the most common cause, though evidence of it is not always present. One of us saw a child eleven years of age who presented the typical signs of an aneurism of the transverse arch.

**Hypoplasia of the Arteries.**—This condition, described by Virchow, has been mentioned in our description of chorea. Virchow sometimes found a small heart, though usually the small aortic calibre is directly responsible for cardiac hypertrophy. The condition has not been recognized during life. It is very rare.

**Embolism** is rare and is almost never seen under five years of age. It is usually caused by particles of vegetations being swept away from diseased heart valves. Septic embolism may occur, however, and Ridlon dwells upon tuberculous emboli in bone and joint tuberculosis. The symptoms depend upon the nature of the embolus and the vessel that becomes plugged. The brain, spleen, kidneys and lungs are the organs that most commonly suffer.

**Thrombosis** is occasionally noted, though not so commonly as in adult life. It is usually a result of some infectious disease and most frequently of diphtheria, typhoid fever, scarlet fever and pneumonia. It has also been observed in the new-born after difficult labor. It has

occurred in the inferior cava in the presence of abdominal tumors and much enlarged lymph nodes. "Chicken-fat" or "current jelly" clots may also be observed in the heart, the former particularly in cases of slow death. Many vessels have been found to contain thrombi; viz., "The sinuses of the brain, the internal jugular, the superior vena cava, the inferior vena cava, the aorta; the femoral veins. The symptoms and physical signs of thrombosis in the infectious diseases are the same as those observed in later life. The prognosis as to eventual recovery in phlebitis of the femorals is much better in childhood. The treatment should be spelt in one word—rest. Elevation of the affected part, when possible is also of value. One of us has seen remarkable results follow the internal administration of citric acid and the external application of collargol in ointment.

**Dilatation of veins** may often be a symptom of local obstruction to the return flow of blood (as in abdominal tumors, hepatic cirrhosis, bronchial gland enlargement). It is much rarer in cardiac disease than in adult life. Hochsinger dwells upon prominence of the facial and thoracic superficial veins in blonde children of the tuberculous type. This we have often seen, and we agree with him that it usually disappears spontaneously.

With venous and capillary nevi (*nævi*) and moles (*nævi pigmentosa*) we shall not deal other than to call attention to their tendency toward sarcomatous change (Keen, etc.). It is well to advise their removal or destruction.

## CHAPTER XI

### BLOOD-PRESSURE STUDIES IN EARLY LIFE

Since the early attempts of v. Basch to study blood pressure by exerting direct pressure over an artery, great advances in method have been attained, and sphygmomanometric estimations have come to mean much in the diagnosis, prognostication and treatment of adult diseases. In view of such amassing evidence, it is singular that similar investigations have not been pursued more generally in studying sick children, and that standard text-books upon pediatrics are practically silent upon this important subject. The literature already presents a number of monographs on blood-pressure studies in infancy and childhood, however, and we are much indebted to Dr. J. F. Prendergast for calling our attention to a number of illuminating articles dealing with various phases of the subject.

Our own researches, though pursued for a relatively short period of time, have still served to show the importance of such studies in meningitis, mental deficiency, hydrocephalus, nephritis, valvular diseases of the heart, pericarditis, pneumonia, typhoid fever, peritonitis, etc. We have come to agree with Gibson that "it is quite as scientific to estimate temperature by the sense of touch, as to try and estimate the blood pressure by tactile sensation."

**Instruments and Method.**—In most of our own work, we have used the "Tycos" instrument. Enclosed in its compact case, it occupies little space in the medical grip. For the pressures encountered in childhood, we have found it quite accurate. Its alleged limitations with high pressures do not concern the student of the child. One fault of the instrument, admitted by the manufacturers, is that the needle does not always return immediately to the zero point, when the instrument has been in use. In general, however, the instruments devised by Dr. Faught seem open to fewer criticisms than any instruments in their respective classes.

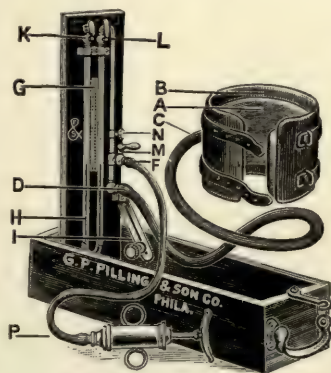


FIG. 43.—FAUGHT STANDARD MERCURIAL BLOOD PRESSURE APPARATUS.

For accurate work with infants and young children, a narrow arm band ("infantile band") is needed.

As a rule, systolic readings are more important in childhood than diastolic readings. Nor is it so important to estimate pulse pressures and mean pressures as in the study of sick adults. We say this because children very infrequently exhibit vascular disease, and increased vascular tonus is a relatively rare phenomena. Nevertheless, it was only recently that a high systolic and low diastolic reading called the attention of one of us to the existence of aortic insufficiency.

To estimate the systolic pressure, the tactile method is usually employed, the reading being made as soon as the obliterated pulse



FIG. 44.—HALF SIZE OF THE INDICATOR IN THE FAUGHT APPARATUS.

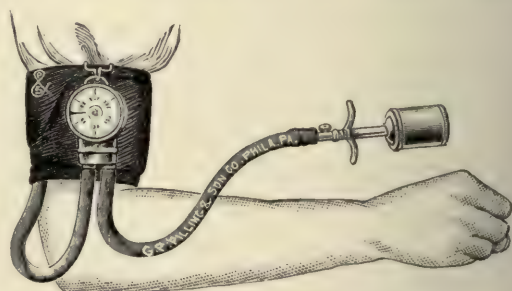


FIG. 45.—FAUGHT POCKET BLOOD PRESSURE APPARATUS (ANEROID) IN USE.

returns at the wrist. In one respect, though, our experience has not accorded with that of others; for we find the pressure obtained in this way to be the same as that obtained by the auscultatory method. In securing diastolic readings, the auscultatory method proves most reliable (the disappearance of vascular sounds).

**Blood Pressure at Different Periods of the Child Life.**—Our present statistics are not large enough to permit us to speak authoritatively upon this important phase of the subject, and we have made far more studies in children than in infants; so we shall quote the figures of others, beginning by placing side by side the well-known finds of Howell and Gordon.

Age	Howell	Gordon
3 years.....	91 mm.	81 mm.
4 years.....	89 mm.	82 mm.
5 years.....	95 mm.	86 mm.
6 years.....	96 mm.	88.5 mm.
7 years.....	102 mm.	85 mm.
8 years.....	101 mm.	93 mm.
9 years.....	102 mm.	100 mm.

Age	Howell	Gordon
10 years.....	112 mm.	95 mm.
11 years.....	102 mm.	104 mm.
12 years.....	111 mm.	105 mm.
13 years.....	107 mm.	
14 years.....	110 mm.	
15 years.....	109 mm.	
16 years.....	117 mm.	
One year.....	73 mm.	
Under one year.....	71 mm.	

In the opinion of Dr. Prendergast, Dr. Gordon probably used a wider arm band than Dr. Howell, which would naturally give him lower readings. As far as our own researches go, we should feel free to say that there is no great disparity here; at least no greater than one may frequently encounter in different children; or even in the same child at different times.

Between one and ten years, Thayer finds an average blood pressure of 110 mm. In early life, Krehl observed pressures ranging from 75 to 90 mm. From eight to fourteen years, Lauder Brunton finds an average pressure of 90 mm.; while from fifteen to twenty years, it ranges from 100 mm. to 115 or 120 mm.

Cook gives as the normal blood pressure

At 6 months.....	75-90 mm.
At 2 years.....	85-95 mm.
Early childhood.....	90-105 or 110 mm.

For an infant of eighteen months, 80 mm. may be considered moderately low; 70 to 75 mm. low and 60 mm. very low.

**Conditions that Influence Blood Pressure.**—Following a plan somewhat similar to that of Anders and Boston, we shall group these various potential factors under three headings: 1. Conditions that raise pressure; 2. conditions that lower pressure; 3. conditions that cause a primary rise followed by a later fall of pressure.

1. **Conditions that Raise Pressure.**—a. Open-air treatment and elevation in altitude. The transference of tuberculous patients from the ward to the open, causes a substantial increase in blood pressure (Hoobler and Hoover). The same change is uniformly noted in the pneumonic patient. Norris believes that we do much for the febrile heart by open-air treatment. High altitudes also induce rises of blood pressure.

b. Moderate exercise should bring about a rise in pressure. If it does not, the heart is weak (Eliot).

c. Cold applied to the surface of the body raises blood pressure.  
d. Emotions, pain and mental effort usually raise blood pressure.  
e. Stimulation. As a guide to the use of stimulants, Cook believes that blood pressure studies are of much importance. In single doses, he finds that alcohol has little effect upon pressure. In repeated doses, it does raise it, however. Strychnine is more reliable, the resulting elevation from an adequate dose lasting from two to six hours. Digitalin, in sufficient dosage, is also valuable; though, according to Cook, its effects do not last so long. Epinephrin may raise pressure to a remarkable degree, so remarkable that it should be administered with caution when the heart muscle is known to be weak. Normal salt solution is of little value for this purpose, unless the subject has suffered loss of blood.

f. Anesthesia. Nitrous oxide raises blood pressure to a marked degree. It should not be employed when there is present a brain tumor or other cerebral condition attended by increased intra-cranial pressure. As children rarely exhibit arterial disease, however, it certainly represents the anesthetic of choice for most operations of short duration. Goodall and Reid state that this increase is almost eliminated when a gallon of oxygen is inhaled prior to the administration of the gas. On the other hand, when oxygen is administered during the anesthesia, the pressure rises rapidly (Faught). Ether raises the pressure primarily; but if pushed to the point of deep narcosis, the pressure falls according to the degree of saturation. No brain surgery should be attempted in childhood without accompanying blood-pressure records. Indeed we have seen enough to convince us that these studies are of invaluable service in all surgical work.

g. Nephritis. In no other disease of childhood is there found "the high range of blood pressure" that we note in this disease (Gordon). The same writer states that the patients with the very high blood pressures have less edema; but they often have blood in the urine. Butterman confirms this statement concerning high pressure in acute nephritis, and finds such studies of great importance in cases of post-scarlatinal nephritis. Blood pressure is also elevated in chronic parenchymatous nephritis. On the other hand, albumin and blood in the urine dependent upon cardiac causes, will be attended by low blood pressure (orthostatic albuminuria).

h. Mental deficiency and epilepsy. In hydrocephalus, the pressure is usually high, sometimes very high. The same is true of microcephalus. During the epileptic seizure, the blood pressure is also very high.

i. Epidemic meningitis and epidemic influenza. In epidemic meningitis, G. Canby Robinson finds increased intra-cranial pressure an almost constant factor. Heightened blood pressure of a moderate degree is not infrequently present in the early acute stages; when exacerbations occur late in the disease, and when the malady takes on a chronic character. The height of the blood pressure bears a relation to the severity of the disease, being high when the symptoms are severe and low in convalescence. No constant result is observed when fluid is withdrawn by lumbar puncture, though there is usually a fall in pressure. He cannot assert positively that heightened intra-cranial pressure induces an invariable rise in blood pressure, unless late in the disease, when internal hydrocephalus has resulted from the blocking of the foramina of exit in the fourth ventricle. Sophian, however, arrives at somewhat more definite conclusions concerning the withdrawal of cerebro-spinal fluid, and also finds blood-pressure studies of great service in determining the amount of Flexner's serum that may be injected with safety. His conclusions are as follows: "1. The old method of administering serum is inaccurate and sometimes dangerous. 2. Blood-pressure change is a very accurate guide to the quantity of serum that can safely be injected, frequently also indicating the quantity of cerebro-spinal fluid that can be withdrawn. 3. The average dose of serum as controlled by blood pressure is smaller than by the old method. 4. Following an injection of serum, controlled by blood pressure, the after effects are usually much more severe."

On theoretic grounds, we feel that Dr. Sophian's interesting results need further confirmation.<sup>1</sup> We have seen one infant die very soon after the injection of the antimeningococcic serum; but on the other hand, it is acknowledged that the serum is a very weak antitoxic agent, and most authorities have been impressed with the need for large dosage. (See Epidemic Meningitis.)

In several cases of epidemic influenza we have observed increased blood pressure during the febrile stage of the disease.

j. In beginning peritonitis and in appendicitis the blood pressure is usually high. Theoretically, one would expect it to be low early in intussusception, though we know of no investigations of this particular point.

k. In lead poisoning, the pressure is invariably higher than normal.

l. Chronic valvular disease. In mitral regurgitation, with compensation, blood pressure is found above the norm. In aortic regurgitation, the systolic reading is higher than normal, while the diastolic

<sup>1</sup> Dr. Flexner's most recent contribution tends to strongly support Dr. Sophian's views.

pressure is low. In this disease, both Hare and Hill have found the pressure higher in the leg than in the arm.

2. **Conditions that Lower Pressure.**—a. It is lower in the weak than in the strong.

b. Bad hygiene, particularly lack of fresh air and bathing, tend to lower pressure.

c. Lowered barometric pressure lowers the blood pressure.

d. Violent exercise, particularly in the untrained. Barach's studies, after a Marathon race, showed an average fall of blood pressure of 20 per cent. In the sturdy this soon rose again to the normal point; but untrained individuals continued to exhibit lowered pressures many weeks and even months after the race. Their hearts also remained dilated. Such races, and indeed all other violent competitive sports should be most carefully supervised. No contestant should be permitted to enter who is not physically strong, whose circulation is not normal, and who has not trained religiously. It is our own firm conviction that pediatricians should go farther than this. That we should place our ban upon all such violent competitive sports in the immature. The American game of football and all long distance running should be prohibited in our leading preparatory schools. An "English Coach," viewing a game on "Soldier's Field," inquired why the finest looking specimens of manhood were standing on the side lines. The Harvard Coach informed him that most of these men had been hurt in preparatory schools. What is true of knees and ankles is also true of hearts. More men would bring perfect physiques to college sports and life's battles, were football and long distance running forbidden in grammar, high and preparatory schools.

e. In shock, blood pressure is lowered to a very great degree. Surgically speaking, this may result from the anesthetic *per se*, or from the undue handling of exposed tissues, particularly intra-abdominal tissues or those in the neighborhood of the larynx.

f. In the phenomena of anaphylaxis and peptone poisoning, the same conditions obtain that are found in shock, and blood pressure is very low. Indeed, the term anaphylactic shock is now commonly employed, and it is recognized that the patient is practically bled into his own abdominal vessels. According to several authorities, among them Crile, the subcutaneous or intravenous injection of adrenalin in normal salt solution represents the most efficient means of combating this condition. Pierce and Eisenbrey recommend an intravenous injection of adrenalin chloride (1-40,000), and also employ the more generally used cardiac stimulants.

g. Chloroform consistently lowers blood pressure in nearly all cases. Ethyl chloride is another dangerous anesthetic. We recently saw a chart exhibiting a fall of pressure from 120 to 80 mm. and yet complete analgesia had not been induced by the latter anesthetic (Courtesy of Dr. Faught). We should again emphasize the importance of blood-pressure studies as routine measures during the administration of anesthetics.

h. In most febrile diseases the blood pressure is low. This is notably true of tuberculosis, typhoid and diphtheria. Even when fever is present in tuberculosis, the pressure is nearly always low. Low blood pressure with dyspnea should make us think of tuberculosis, just as high pressure with dyspnea should direct our attention toward the possibility of nephritis. In general, typhoid fever presents a low blood pressure, though in several children ill with this disease, we have found it quite normal during the early days of the disease, but invariably falling during the stadium of the fever. Very low pressures in typhoid fever, particularly such as fail to respond to stimulants, are of dire significance. In one of our recent cases, a pressure of 70 enabled us to predict and to prepare for some of the serious phases that his disease subsequently took. Diphtheria lowers blood pressure more surely than any other infectious disease. On the other hand, in two recent cases, we have observed a rise to the normal point after the administration of antitoxin. Of course, we would not draw conclusions from so small an experience, but the finds were so unusual for diphtheria, that we note them in passing.

In our own minds, there is little doubt that death in infectious disease often occurs from paralysis of the vaso-motor center. The term hypoadrenia probably well describes the etiology of such low pressures. During convalescence from nearly all infectious disease, the blood pressure is low.

i. Jaundice lowers blood pressure, just as surely as it diminishes the bodily temperature, and slows the rates of respiration and the heart. This observation has proved of importance in the study of the jaundice of the new-born.

j. Cardiac and pericardial disease. In myocarditis, acute endocarditis, and acute pericarditis, blood pressure falls. With a large pericardial effusion, it may fall to a very dangerous level. It is also low in failing compensation. Indeed we have failed to find any evidence of pressure in one case, just as we failed to detect a pulse at the wrist. In auricular fibrillation it is irregular, different systoles

exhibiting different levels. In mitral stenosis and aortic stenosis, the pressure is low.

k. Loss of blood naturally induces a fall in blood pressure. It is in these hemorrhagic cases that the injection of normal saline or other fluid may serve to reestablish the pressure. One-fifth of the total quantity of blood may be lost, and yet there may be sufficient pressure for life, simply because the increased constriction of the peripheral vessels is sufficient to bring about the desired result. If blood volume is reduced to one-half, convulsions and death will ensue (Stowell).

l. Pleural aspiration causes a fall in blood pressure, sometimes to the point of danger. It is unwise to irritate the pleura with instruments, etc.; long drainage tubes should not be permitted to protrude into the pleural cavity, and irrigation of the pleura should never be performed (Capps and Lewis).

m. Lumbar puncture and the injection of Flexner's serum. These matters have been dealt with in the consideration of blood pressure in epidemic meningitis.

n. In neurasthenia blood pressure is low.

o. It is lowered in most cases of mental deficiency. The exceptions, already noted, are microcephalus, hydrocephalus, and in epilepsy during the convulsion.

p. A distended stomach or intestine may interfere mechanically with heart action, and so the blood pressure may fall.

q. Iodides lower pressure, provided that structural changes in the vessels are absent.

**3. Conditions that Induce a Primary Rise, Followed by a Later Fall in Blood Pressure.**—We have spoken of a number of conditions that produce these results, including violent exercise, and epidemic meningitis; but the affections that almost invariably induce this sequence, and that do so to notable degrees, are acute peritonitis (including appendicitis), scarlet fever and pneumonia. In all three the blood pressures are high in the early stages, though in all three they may fall to very dangerous levels later on in the disease.

## CHAPTER XII

### DISEASES OF THE BLOOD

#### GENERAL CONSIDERATIONS AND DEFINITIONS

**The Total Amount of Blood.**—In the adult the amount of blood in the body is said to equal  $1/13$  of the body weight; in the infant the total quantity of blood is said to be but  $1/19$ – $1/20$  of the body weight. Infants stand hemorrhage badly.

The essential features for consideration in a study of the blood are its coagulability, its hemoglobin content, its specific gravity, and the red and white corpuscles. Our knowledge of the so-called blood plaques or plates is as yet too limited to warrant more than a reference to their existence in this chapter.

In adult health a uniform and recognized ratio is usually maintained between the number of the red and white cells, although slight discrepancies occur in all counts made of the same specimen by different observers. For all practical purposes the normal number of red cells is estimated at 5,000,000 in a cubic millimeter, and of white cells at 7500 in a cubic millimeter, but the latter may vary in health from 5000 to 10,000. Coles says that Hayem places the number at 6000 and von Lembeck at from 8000 to 9000. The proportion of white to red cells in normal blood is as 1 to 500 or 600. For clinical and diagnostic purposes a careful study of the various types into which the white corpuscles are divided is of the greatest importance. In early life, however, the blood possesses different characteristics, qualities that if observed in later years, would be considered pathologic. For convenience sake, we shall approach this study at three periods of life: 1. At birth; 2. in infancy; 3. during childhood.

1. **The Blood at Birth.**—Japha, quoting from the most recent studies says, the blood of the new-born infant presents the following characteristics: a. A high specific gravity (1060–1080); b. a high percentage of hemoglobin (100 per cent. to 140 per cent.); c. a very large number of red cells (5,825,000 to 7,550,000); d. an increased number of leukocytes (as high as 36,000(?)); e. a preponderance of polynuclear (polymorphonuclear) cells (73.4 per cent.); f. nucleated red cells up to the third day. All of these differences are marked up to

the fourth day of life. By the middle or end of the first month, however, the infantile characteristics are present.

2. **The Blood During Infancy.**—a. A lessened percentage of hemoglobin (65 per cent. to 80 per cent. (Holt)). b. Fewer red cells than in the adult. c. From 12,000 to 13,000 leukocytes to the cubic centimeter (often more). d. A preponderance of lymphocytes (50 to 55 per cent. according to Japha). They also exhibit greater disparity in size than in adult life and the larger forms are more numerous. e. Transitional forms of leukocytes are more common. f. Myelocytes and normoblasts are but rarely found.

3. **The Blood in Childhood.**—From the infantile period to the fourteenth or fifteenth year, the blood gradually acquires its adult characteristics. Leukocytosis may occur from relatively slight causes, however, throughout this period of existence.

It is very essential for the student to understand the few facts that follow, to enable him to differentiate the separate ages or forms of the white cells should he desire to make a blood examination for diagnostic purposes. The youngest white cell is the "lymphocyte," and the oldest is the "eosinophile," and the "polymorphonuclear" or "neutrophile" is the intermediate. The lymphocyte is the simplest and smallest, as well as the youngest, and consists of a small amount of protoplasm with a large nucleus. It measures from  $6\frac{1}{2}$  to  $7\frac{1}{2}\mu$ . The neutrophile is made up of a large proportion of protoplasm which has already become granular, and an irregularly outlined nucleus, often horseshoe-like, always indented, more frequently presenting the appearance of many nuclei (possibly as many as five) entirely free from one another, but usually, upon closer examination, they will be found connected by small trabecular bands.

The neutrophile is larger than the lymphocyte, measuring from  $7\frac{1}{2}$  to  $9\frac{1}{2}\mu$ . The eosinophile is more highly granular, the granules being larger and more refractile than in any other normal cell, its nuclei being similar to that of the neutrophile. The size of this cell is from 8 to  $9\frac{1}{2}\mu$ . Often white cells are observed which possess morphologically the same characteristics as the lymphocytes, but they are much larger, measuring from  $8\frac{1}{2}$  to  $12\mu$ , and are simply named the "large" lymphocytes. A modification of this cell is one in which the nucleus becomes indented or kidney-shaped and its protoplasm shows a tendency to become granular. It is known as the "transitional cell." These varieties of cells bear certain numerical relations to one another, although different observers give different proportions, as will be seen from the accompanying table:

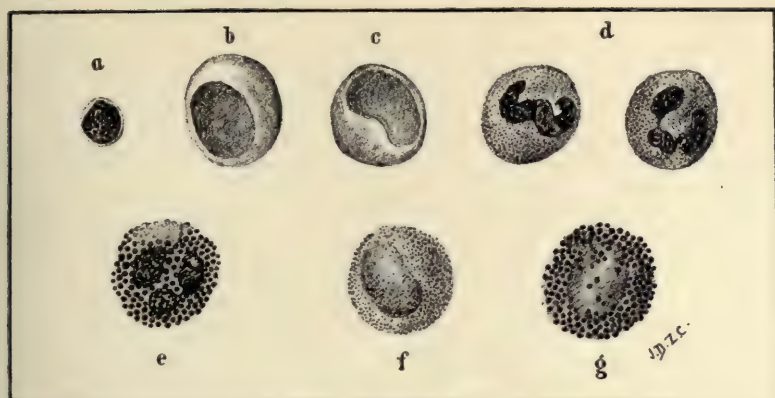


FIG. 46.

a. Small lymphocyte. b. Large lymphocyte. c. Transitional lymphocyte. d. Neutrophiles.  
e. Eosinophiles. f. Myelocyte. g. Eosinophilic myelocyte.

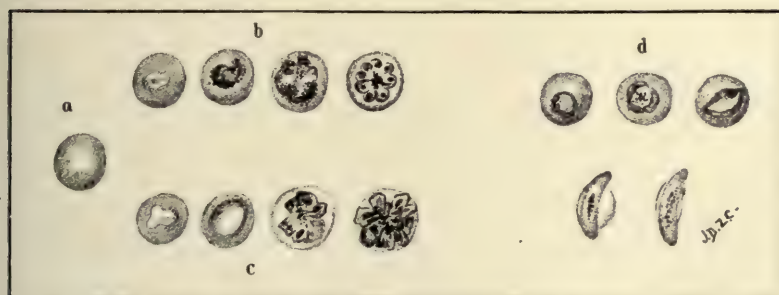


FIG. 47.

a. Normocyte. b. Parasites of quartan fever. c. Parasites of tertian fever. d. Parasites of estivo-autumnal fever.

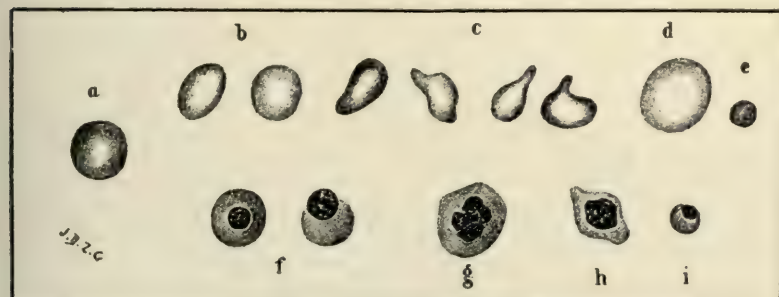


FIG. 48.

a. Normocyte. b. Normocyte deficient in hemoglobin. c. Poikilocytes. d. Macrocyte.  
e. Microcyte. f. Normoblasts. g. Megaloblast. h. Poikiloblast. i. Microblast.

	Cabot. Per cent.	Rotch. Per cent.	Stengel. Per cent.
Lymphocytes, small.....	20 to 30	24 to 30	25
Lymphocytes, large.....	4 to 8	3 to 6	3 to 6
Neutrophiles.....	62 to 70	60 to 75	65 to 70
Eosinophiles.....	0.5 to 4	1 to 2	Not over 3

It will readily be perceived that these figures relate to adult blood. Gundobin's figures (quoted by Koplik) come much closer to what one usually finds in hematologic investigations in infancy.

	Polymorphonuclear leukocytes	Mononuclear lymphocytes	Transitional forms
Immediately after birth....	63 per cent.	25 per cent.	12 per cent.
Forty-eight hours after birth.	70 per cent.	21 per cent.	19 per cent.
Infancy.....	34.6 per cent.	59 per cent.	6.4 per cent.

The red cells, also called erythrocytes, are biconcave discs, smooth, homogeneous, and without a limiting cell membrane. In health three sizes of these corpuscles are found, the larger ones, called "macrocytes" or "megalocytes," measuring from 8 to 9 $\mu$ , which constitute about 12 1/2 per cent. The medium-sized corpuscles have an average diameter of 7 1/2 $\mu$ , and constitute about 75 per cent. of the entire number. The small corpuscles, called "microcytes," measure from 6 to 6 6/10 $\mu$ , and constitute about 12 1/2 per cent. (See Fig. 45.)

The study of the blood for clinical purposes is greatly facilitated by means of various stains. Multiple staining is practically always employed. Many workers prefer the double stain of eosin and hematoxylin or eosin and methyl-blue, while others use the triple stain, after the method of Ehrlich, the formula for which is as follows:

Saturated watery solution of orange G.....	120 to 135 c.c.
Saturated watery solution of acid fuchsin.....	80 to 165 c.c.
Saturated watery solution of methyl-green....	125 c.c.
Glycerin.....	100 c.c.
Absolute alcohol.....	200 c.c.
Distilled water.....	300 c.c.

Eosin solutions are made to the strength of saturation, and the

hematoxylin solution is prepared according to the formula of Delafield, which is:

Crystallized hematoxylin.....	4 c.c.
Absolute alcohol.....	25 c.c.

Mix and allow to stand two days, then add 400 c.c. of concentrated aqueous solution of ammoniac alum, stand aside for four days, leaving it uncorked and exposed to the light. Filter and add 100 c.c. of glycerin and 100 c.c. of wood-alcohol. This solution ripens in three to four months, and for careful work should not be used until it has been prepared for that time. For staining purposes a drop of blood is secured from the well-cleaned finger-tip or lobe of the ear upon a scrupulously clean cover-glass. Whichever site is selected should be carefully washed with soap and water, afterward with alcohol or benzin. A second cover-glass is quickly placed over the first, and by rapidly sliding them apart two smears are secured, which are dried by exposure to the air. An hour will suffice, although they can be kept indefinitely in this condition, and especially so if they be protected from air, dust, and moisture. The staining process is very simple. Heat the smears for about an hour upon a brass plate raised to such a temperature that a drop of water will evaporate rapidly from its surface, then stain for five minutes with a hematoxylin solution, which is then thoroughly washed off in running water, and the film afterward allowed to stand for two or three minutes in water. It is then again stained with eosin, and after drying is ready to be mounted in balsam. An oil-immersion lens should always be used for blood work.

**General Pathologic Changes.**—Before the study of hematology, the blood was considered, pathologically, solely from the clinical evidences of an increased or diminished quantity of the fluid in relation to the bodily weight. Little attention was paid to the quality or to the condition of the various constituents. *Plethora* was described as a condition in which there existed an excessive quantity of blood, and while it seems evident that a transient increase in the total amount of blood is a possibility, yet the condition is purely relative, and the symptoms observed are the result of vasomotor disturbances. The opposite condition to plethora was indicated by the term *anemia*, which was held to represent, in a general way, an impoverished state of the blood. *Oligemia* is the term used to express an actual reduction in the amount of blood without any reference to component parts. *Oligocythemia* expresses a decrease in the number of red blood-corpuscles, and *oligochromemia* expresses the diminution in the amount of hemoglobin

in the blood. *Hydremia* expresses the fact that the watery element of the blood is increased beyond its normal amount, and *anhydremia*, a decrease in the same element. *Lipemia* expresses an excess of fat in the blood and is absolutely of no clinical significance, although it is found in a number of conditions, such as phthisis, nephritis, diabetes, etc. *Melanemia* refers to pigments in the blood and occurs in some infectious diseases, but especially in malaria, in which disease it may be free in the blood after a paroxysm, although the pigment is normally contained in the corpuscles.

An increase in the number of the white cells may affect all varieties of these cells, but especially the lymphocytes and neutrophiles, constituting a condition known as *leukocytosis*. A physiologic increase of the white cells is at times observed, and especially is this liable to occur after eating, an hour or so after which the white corpuscles may be increased to 9000 or 10,000 in a cubic millimeter. In infants this is especially marked. A pure lymphocytosis is very rarely seen and is said to exist when the number of white cells reaches 10,000 in a cubic millimeter, and consists of an increase of the lymphocytes.

*Leukocytosis*, as a physiologic condition, exists in pregnancy, as a result of postpartum hemorrhage, after violent exercise, a cold bath, and massage. The moribund state is also accompanied by a leukocytosis. Pathologically, we are confronted by leukocytosis most frequently after all inflammatory conditions, and a general law may be laid down that the leukocytosis bears a direct relation to the severity of the infection and to the powers of resistance of the individual. In the weak and emaciated, with low resisting power, a severe septic infection will not cause such a degree of leukocytosis as a similar condition would give rise to in the strong and vigorous. Should the disease be very acute and spreading, the leukocytosis is more marked than when it has become limited and walled off. This has been more thoroughly studied in appendicitis than in any other disease. Cabot has carefully tabulated many diseases during the course of which a leukocytosis is found. Among children it is most frequently seen in the infectious diseases: scarlet fever, diphtheria, follicular tonsillitis, pneumonia, smallpox, cerebrospinal meningitis, whooping cough, and among the septic diseases, appendicitis, abscesses of all regions, except those of tubercular origin, and many forms of skin diseases. When leukocytosis is absent in croupous pneumonia, it is a very bad prognostic sign. Many diseases that produce toxemias also produce leukocytosis. It is, however, normally absent in typhoid fever, influenza, malaria, uncomplicated measles, and almost all tubercular con-

ditions, so long as there is not a mixed infection. Indeed, in typhoid fever and influenza, leukopenia may be observed. In the former disease, we regard its presence as strong confirmatory evidence.

There are certain intrinsic changes in the red blood elements which are important in diagnosis. An increase in the proportion of small red cells is called *microcythemia*, and an increase of the large cells is called *macrocythemia*. The importance of microcythemia has not been clearly made out, while macrocythemia is associated with all the severe anemias. When any alteration occurs in the size of the corpuscles, there are usually found associated changes in their forms. Poikilocytosis is the term used to designate all these changes in shape. Corpuscles may be rod-shaped, pear-shaped, etc., assuming various forms, in the place of the uniformly round body. Occasionally all the cells may appear uniformly but irregularly outlined, owing to faulty technic.

Red blood-corpuscles only stain as they die, and normally take an acid stain (eosin), but in a number of diseases, such as severe anemia, scarlet fever, and smallpox, a few red corpuscles take on in parts the second stain. This condition is called *polychromatophilia*, and is an evidence of cell degeneration. One observer considers it an evidence of cell death. Red corpuscles may show small knobs projecting from the spheric border, which become detached and float in the plasma. Red blood-corpuscles in disease take on the fetal phenomenon of *nucleation*. Nucleated corpuscles floating in the peripheral blood are comparatively rare, and it may require many examinations of the same specimen to find them. *Nucleated corpuscles* are of three sizes: the normoblast, the microblast, and the megaloblast. Normoblasts occur normally in bone-marrow, and are regarded as immature red cells. Their presence in the circulation always points to such blood changes wherein the demand for new material has been greater than the supply, and where raw material had to be drawn from. On the contrary, a megaloblast is always foreign to the healthy body, and is only found pathologically and in the fetal marrow. Its presence is indicative of a severe anemia. In size it is always over  $10\mu$ , and, according to Cabot, may be as large as  $20\mu$ . It is often polychromatophilic. The nucleus is very large, staining less intensely than a normoblast, and, as a rule, uniformly. When present, it is an evidence of a return to the fetal hemogenesis, and that the bone-marrow has become very seriously deranged in its function of blood formation. Megaloblasts may also be poikilocytes. Microblasts are smaller than the ordinary red cells, and have probably no significance other than that of normoblasts or

megaloblasts. In general the nucleated red cells are evidences of severe trouble, and occur both in primary and symptomatic anemias. Coles says that normoblasts may always be looked for when the red cells do not exceed 2,500,000 in number. However, it should be stated that the anemia following hemorrhage must be very severe before nucleated red cells are found. At any time when the megaloblasts exceed the normoblasts the condition of the patient is very serious, and especially is this true if the megaloblasts are also poikilocytes. Red cells may sometimes contain granular and hyaline masses, the result of the parasites of malaria, which will be described when speaking of that disease.

Myelocytes, or marrow-cells, never occur in normal human blood, but they are very common in the red bone-marrow and are characteristic of mixed and splenomedullary leukocythemia. Their diameter may reach  $20\mu$ . The nucleus is usually single and faintly stained, occupying a very large part of the cell, the remainder of which is highly granular. Occasionally myelocytes are small, and were it not for the fact that the granules take up certain stains, such as Ehrlich's neutral stains, they could not be distinguished from large and small lymphocytes. These granules do not stain with hematoxylin. The nucleus is often lobed. Myelocytes contain at times eosinophilic granules, and are then called eosinophilic myelocytes. The chief changes in the white cells are in their numbers and proportions, which are determined by the differential count.

The granules found in the white cells are of five varieties: Those which are stained by the acid stains and are called "eosinophiles," the granules of which are large and round; these exist in the normal blood. "Amphophiles" contain small round granules which take both acid and basic stains and do not normally exist in the human blood, but in the medullary cavity of bones and in the leukocytes of some of the lower animals. The so-called "Mast-cell," which is a basophile—that is, takes a basic stain—contains coarse, round, and poorly refracting granules. Ehrlich believes they do not occur in the blood normally, but if found in any number, are pathognomonic of leukocythemia. Ordinary stains do not show the granules, but they can be readily brought out by the use of the following solution, in which the films should lie twenty-four hours:

Dahlia, saturated alcoholic solution.....	50 c.c.
Glacial acetic acid.....	10 to 15 c.c.
Distilled water.....	100 c.c.

The granules may also be stained with methyl-blue. The next

variety is only distinguishable by the size of the granules, which are smaller and are also spoken of as Mast cells. Both of these cells are mononuclear, measuring about  $20\mu$ . "Neutrophiles" are those cells in which the granules take only the neutral stain. The granules are fine, and fill up a large portion of the protoplasm of the cells. In regard to both varieties of the Mast cells, there is at present a great deal of doubt in the minds of different observers. The eosinophilic granules he calls  $\alpha$ , the amphophilic  $\beta$ , the two varieties of basophilic  $\gamma$  and  $\delta$  respectively, and the neutrophilic  $\epsilon$ .

The **instruments used in the inspection of the blood** are the microscope, the hemocytometer, the hemoglobinometer (several improved forms of which have recently been devised), the hematocrit, and the spectroscope.

Within recent years the literature upon this fascinating subject has been richly increased, and the reader is referred to that for more detailed descriptions of the instruments used and the technic employed, which cannot be given in this chapter.

## ANEMIA

Anemia is divided into two classes, primary and secondary. First, the class of primary anemias containing all those disorders which are essential and in a direct manner related to or dependent upon the blood-making organs, and, so far as we know, bear no relation to any extraneous causes. This class includes chlorosis, leukocythemia, pernicious anemia, pseudoleukemia infantum, and Hodgkin's disease.

Second, the secondary or symptomatic anemias, which are the result of some other distinct disease or derangement of function and are not due to disorders of any of the organs or tissues directly concerned in the blood-making function. Hemorrhage from any cause presents the most perfect example of a secondary anemia. It is also found to exist in association with, and after, acute fevers, infectious diseases, tubercular diseases, syphilis (acquired and hereditary), rachitis, malignant growths, and especially in children after gastric or intestinal diseases. Marfan believes that primary anemias do not exist in nurslings, and that these and the enlargements of the various organs

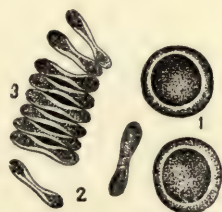


FIG. 49.—HUMAN COLORED BLOOD-CORPUSCLES.—(After Landois.)

1. On the flat. 2. On edge. 3. Rouleau of corpuscles.

taking part in the production of blood depend upon some acute or chronic infection. He also believes that hypertrophies of the spleen, liver, and lymphatic glands are very prone to appear in any of the anemias of childhood. However this may be, it is certain that in many secondary anemias the blood of the infant or young child may present changes that are only seen in the primary anemias of adult life (poikilocytes, normoblasts, etc.).

In all forms of anemia the so-called hemic murmur may be found. However, in the anemia of children these inorganic murmurs are uncommon until after the third year. In all anemias of children, and particularly of infants, the changes found in the blood are much greater than those under similar conditions in the adult. These changes consist of a nucleation of the red corpuscles and in an increase in the proportion of the lymphocytes, associated at times with irregularly shaped red cells.

### PRIMARY ANEMIAS

#### Chlorosis

**Synonyms.**—CHLOREMA; GREEN SICKNESS

**Definition.**—Chlorosis is an essential anemia occurring chiefly in young girls at about the period of adolescence. It is characterized by a diminution in the percentage of hemoglobin, the various evidences of anemia, changes in the vascular system and lack of development of the genitalia.

**Etiology.**—Among the predisposing factors which are potent in the causation of this disease sex stands preeminent. Chlorosis seems to be confined chiefly to the female sex, and is rarely seen in other than ill-nourished girls about the age of puberty, although cases have been reported in females before reaching this period by Nonat and others. Chlorosis exists in boys in a somewhat modified form. Heredity undoubtedly plays an important part as a predisposing cause. The statement that light-haired girls of poor vitality are especially predisposed to chlorosis has been generally refuted, although the question is still unsettled. Virchow called attention to narrowing of the aorta in subjects of this disease, and believed it might play an etiologic rôle. This was sometimes associated with a small heart. The theory of Clark probably deserves more attention. He found constipation nearly always present, and believed that toxins from the intestinal canal were responsible for the condition. The causes which may appear directly responsible for the development of the disease are, on one hand, over-

work and poor nourishment, and, on the other, indolence, vicious habits, and bad sanitation. Powerful nervous impressions may act as strong influences in the development of the disease. Many have looked upon chlorosis as dependent upon disturbances of menstruation; but these are probably secondary and not causative. So far the causative element has not been demonstrated, but the consensus of opinion is that chlorosis is dependent upon imperfect nutrition and sanitation.

**Pathology.**—The heart and blood-vessels in chlorosis may be in a state of hypoplasia. On the other hand, the heart is often found dilated. This is particularly true of the right heart. Fatty degeneration of the cardiac muscle and arterial coats has frequently been demonstrated. The same condition of hypoplasia is often seen in the genital organs, they frequently presenting an infantile appearance. Ulcer of the stomach has been mentioned. Tuberculosis (probably secondary) is also occasionally met with. While any or all of the foregoing changes may be noted, they are by no means constant.

**Blood Changes.**—The specific gravity of the blood in chlorosis is low, being often only 1035. The lowered specific gravity is proportionate to the diminution in hemoglobin, readily demonstrated by the von Fleischl or Dare instruments or by the convenient Tallquist papers. The color of the blood as it flows from a prick wound is light. Coagulation takes place rapidly, and manipulations therefore must be rapid. The blood is obviously thin and watery.

The essential change in the blood is a decrease in the actual amount and percentage of hemoglobin present in each corpuscle. There is probably no diminution in the total amount of the blood. The reduction of the hemoglobin occurs with or without a material decrease in the red corpuscles, while in the severer cases they are decidedly decreased in number. Should the case be put upon the usual treatment of iron and arsenic and observations frequently made, the red blood-corpuscles may be found increased in number and the amount of hemoglobin stationary, or nearly so. The color index is always low, being from 0.5 to 0.35 or even to 0.3.

The average amount of hemoglobin in chlorosis is about 40 per cent. Out of 247 cases mentioned by Coles, as tabulated by various writers, 40 per cent. showed a blood count of 4,000,000 corpuscles, and 60 per cent. of less than that number. It is rare to find a very low count, such as occurs in pernicious anemia. The diminution of the hemoglobin is found after certain diseases, while the blood is being regenerated, but not to the same degree as in chlorosis. Certain changes take place in the red corpuscles. Microcytes exist and may be so

numerous that the general average of the diameters of the blood-corpuscles is reduced. The corpuscles may be changed in shape, but the degree of poikilocytosis is not dependent upon the decrease of the red cells.

In staining specimens many red cells fail to take on the average amount of staining. Polychromatophilia exists, demonstrating the mortal changes in the cells. The occurrence of nucleated red cells has been denied by some writers, but the positive evidence of others would seem to settle the matter. They occur only in very severe cases and are usually normoblasts, while the occurrence of megaloblasts in the most extreme cases is rare.

In all uncomplicated cases of chlorosis the white cells remain about normal. There is never a leukocytosis, though there may be a diminution of the white cells. Rarely a few myelocytes are found. Cabot records a table of white-cell counts in which the highest count was 15,000 and the lowest 1500; the average was 7485. In those cases where the count was high there was some other condition prevailing.

**Symptoms.**—The premonitory symptoms of chlorosis are varied, sometimes rather vague. Frequently they are unnoticed, except that the patient, before in good health, develops a gradual increase in lassitude, which is often attributed to the onset of puberty or the effect of overwork. Later there are menstrual irregularities. Shortness of breath and palpitation of the heart are complained of frequently. Headache is common; in fact, in some cases may be almost constant. Dizziness and weakness, increased upon standing and walking, are usually noticed. The digestion becomes weak, the appetite fails, and the patient often craves abnormal articles of food. The skin develops a peculiar greenish-yellow tint which is eminently characteristic of the disease. The mucous membranes become pale, and the conjunctivæ in severe cases almost colorless. In some cases the cheeks and lips retain their natural color, even though the hemoglobin shows a pronounced reduction. To this class of cases Wendt has applied the name of chlorosis florida or chlorosis rubra. Occasionally marked pigmentation is observed in the neighborhood of the joints. Edema beneath the eyes and of the malleoli is commonly observed, and oftener toward the end of the day than in the morning. There is a tendency to the accumulation of fat, making the patient, as a rule, rather flabby than emaciated. The disturbances of circulation are generally manifest by visible pulsations of the veins of the neck. Coldness of the extremities and palpitation of the heart are very frequently complained of. The pulse is generally rapid and weak. Examination of the heart seldom

reveals any change in its size. The apex-beat is usually visible and strong; hemic murmurs are heard upon auscultation, most frequently over the pulmonary area, and at times a soft systolic bruit may be elicited at the apex. Not infrequently a hemic murmur will be heard over the right jugular vein. This is sometimes known as the *bruit de diable*, or "humming-top" murmur. Hemorrhages are not uncommon, and may be due to degeneration of the arterial coats or to blood changes. The nervous symptoms are many and varied; cephalalgia and neuralgia are rarely absent, while hysteric manifestations of a varied and sometimes grave character are not at all uncommon. Optic neuritis and neuroretinitis have been recorded as symptoms by Gowers.

**Complications.**—A large number of cases of chlorosis present no complications. Occasionally, however, endocarditis and enlargement of the thyroid gland are seen. Thrombosis of the veins was first observed by Trousseau, and occurs more frequently than is generally supposed. Gastric ulcer, nephritis, and phthisis are occasional complications.

**Diagnosis.**—Anemia in a young woman with cardiac, digestive, and menstrual irregularity, a greenish-yellow tint of the skin, certain nervous disturbances associated with a diminution of the hemoglobin, very little changes in the blood count as compared with the decrease in the hemoglobin, render the diagnosis easy. We are able in no instance to depend upon the blood examination alone for the diagnosis, or we should be confusing it with syphilis, tuberculosis, malignant diseases, and other diseases which present secondary anemia. (Chloro-anemias.) Leukocytosis is very common in secondary anemia and absent in simple chlorosis.

**Prognosis.**—The prognosis of chlorosis is favorable. There are exceptional cases, however, which resist treatment. Chlorosis is generally amenable to treatment in from six to eight weeks, but sometimes it is most obstinate and prolonged.

**Treatment.**—In chlorosis, as well as in the various symptomatic anemias, special attention should be given to the environment of the patient. In many cases the rest cure or one of its modifications will be found necessary. This method of treatment is particularly applicable to those patients whose lives have been spent at hard work in a close atmosphere. For those whose means will allow it, a sea voyage is to be recommended, and in cases where this luxury cannot be had, quiet recreation in a neighboring park or several hours spent in the sunshine in a public square should be insisted upon. Medicinally, iron can be claimed as a specific in the disease, and of all the forms of iron which

we have at our command, Blaud's pills, containing equal parts of the dry sulphate of iron and the carbonate of potassium, have probably given the most satisfaction. Where, for any reason, pills cannot be taken by the patient, the powdered saccharated carbonate or some liquid preparation of iron should be used. The tincture of the chlorid will often be found extremely beneficial. During a long course of iron treatment constipation will sooner or later result, and therefore the administration of the drug should be so regulated as to avoid this objectionable feature. The natural iron waters are here of special use. Constipation can generally be overcome by the occasional use of small doses of a laxative—a saline, cascara, senna, or bowel irrigation. Next in efficacy to iron in the treatment of chlorosis is arsenic. The good results of the latter are much increased in many cases by combining arsenic with iron. In most cases massage will be found an admirable adjunct in the treatment. A powerful agent in combating the effects of anemia is systematic deep breathing and respiratory gymnastics.

## Leukocythemia

**Synonyms.**—LEUKEMIA; WHITE BLOOD

**Definition.**—Leukocythemia is a primary or essential anemia, characterized by excessive increase in white blood-cells and by an enlargement of the spleen or lymphatic glands, with changes in the bone-marrow.

**Etiology.**—Leukocythemia may occur at any period of life from infancy to old age, and although comparatively a rare disease, particularly in early life, it is as common among the rich as the poor. The infectious origin of the disease has been urged ably by many good observers, but no proof is exhibited as yet.

Among the predisposing causes heredity has commanded considerable attention. Instances are recorded bearing proof of the importance of this factor in the development of the disease. Leukemia has been frequently met with after severe attacks of malaria. However, the latter disease is looked upon as a predisposing rather than an exciting cause. Syphilis, rachitis, typhoid fever, and severe hemorrhages have all been mentioned as predisposing causes. The exciting cause of the disease has not yet been determined.

**Pathologic Anatomy.**—Leukemia is essentially a disease of hemogenic alterations in the blood-making organs. The lymphatic

structures are principally involved, and pathologic lesions are noticed in the spleen and bone-marrow.

From this fact three varieties of leukocythemia have been described: a splenic, a medullary, and a lymphatic. However, since pure splenic cases and medullary cases rarely, if ever, occur, though there is no doubt that one or other of these elements may at times predominate, it is wise to outline but two classes of leukocythemia. This is especially noted in the works of the later writers, who declare that the splenic and medullary varieties are always blended and that even the lymphatic variety may be associated with the other two. The divisions of leukocythemia, then, are first the splenomedullary, in which we find the spleen enlarged, congested, and infiltrated with leukocytes, and the bone-marrow of the long and spongy bones of a yellowish hue, either in small patches throughout the marrow or uniformly present. This material may be firm at first, though in time it softens down and eventually much resembles pus. The fat has been replaced by a purulent or, as Newman says, a pyoid, material. The microscope shows both varieties of marrow-cells, and on account of the very large number of cells showing karyokinesis and in the process of division, it is evident that proliferation has been going on. The nucleated red cells lie along the periphery of the marrow. The occurrence of these cells in the blood is explained by the escape of all these elements from the bone-marrow into the blood. The cause of this condition has not yet been determined.

Second, lymphatic leukocythemia is characterized by the enlargement of the lymphatic glands and the substitution of lymphoid tissue for the bone-marrow. This lymphoid tissue consists of small mononuclear lymphocytes and some nucleated red cells. The spleen in this variety also shows some enlargement. The lymphatic glands are not always enlarged, nor does the spleen always appear to take a minor part, since in rare cases it is quite large. The essential feature is an excessive proliferation of the lymphocytes. The cause of this condition is not known. In this disease the white cells show a very appreciable increase when examined by the hematocrit.

*The Blood in Leukocythemia.*—There are certain facts about the blood that apply to both forms of leukocythemia. Its color is usually normal, though when the white corpuscles are very numerous, it is described as chocolate, but it sometimes looks like pus and blood mixed, or it may be very pale. Its specific gravity is low, its reaction is alkaline, and it coagulates readily. Under the microscope, without stain, the disproportion can be seen between the white and red cells,

and if it be a splenomedullary case, the large size of the white cells can be noted. The leucocytes are enormously increased in number, sometimes as many as 500,000 being found, and a count of 100,000 to 200,000 is not unusual. The proportion of white to red may be 1 to 12, while that in health is 1 to 500 or 600. A few remarkable cases have been reported where the proportion was 1 to 2. It is important to understand that it is not the actual number or proportion of the white cells present which makes the diagnosis, but that it is the character of the cells. A leukocytosis exists oftentimes to a very marked degree, while the normal ratio of the different varieties of white cells remains; in other words, it is quality we look for and not quantity alone. In leukemia, if any intercurrent and infectious disease should arise, the leukocytes grow fewer in a remarkable degree.

Splenomedullary leukocythemia is the commoner of the two forms. Should one not have at hand a case of the disease to study, it can very readily be worked up from the bone-marrow of a kitten mixed with its ordinary blood. The alterations are in the forms and varieties as well as in the number of the leukocytes. The blood should be stained for study. The characteristic cell is the myelocyte. The existence of the myelocyte in a blood-stain will not establish the diagnosis, but when these cells exist in larger numbers, then it would be just to make this diagnosis—30 to 50 per cent. of all leukocytes seems to be the average number. The neutrophiles are very much decreased in relative numbers, though the actual number of these cells present may be enormously increased. The differential count shows about 50 per cent. of neutrophiles, occasionally dropping as low as 15 to 20 per cent. They are smaller and more irregular in shape than in health. The eosinophiles are increased, averaging, according to Cabot's table, 4.4 per cent. In one case there was 11 per cent.

There are three classes of eosinophiles present: First, the ordinary polynuclear eosinophile; second, a small eosinophile that is deeply stained with eosin and that does not occur in the human blood, and which has been thought to be characteristic of this state; third, the eosinophilic myelocyte, which never occurs in health and is characteristic of this disease. The lymphocytes, as a rule, are much reduced in their proportion, being often as low as 5 per cent. in the place of from 15 to 25 per cent., as in health. Karyokinetic figures may be found.

The number of red cells is not extremely diminished, being reduced from two to three millions. The shape and size remain about the same. Nucleated red cells are constantly found in leukocythemia, and it is

said that this is the only disease in which they occur in such numbers. They are normoblasts, with an occasional megaloblast. The hemoglobin is decreased in proportion to the decrease of the red cells. Blood plates are increased, and Charcot-Leyden crystals are found in dried specimens.

*Lymphatic Leukocythemia.*—This form is rare and more acute than the preceding disease; it is characterized by an increase in the size of the lymphatic glands and the occasional enlargement of the spleen, and by the following blood changes: There is an increase in the white cells which is never so extensive as in the splenomedullary type, ranging about 150,000 cells in a cubic millimeter, though there may be as low as 40,000 or as high as 450,000. Lymphocytes predominate, and may form as much as 95 per cent. of all of the white cells. In no other disease does such a state constantly exist. The lymphocytes are large and small, depending upon the case. A few myelocytes may be found. Red blood-corpuscles and hemoglobin are diminished, and nucleated red cells are rare.

**Symptoms.**—Leukocythemia usually comes on so insidiously that the patient does not become aware of his condition until the disease is fairly well developed. Infancy and early childhood present exceptions to this rule, however, for a number of cases of acute leukemia have been reported. The early symptoms are those of a developing anemia, with its attending phenomena of weakness, cardiac palpitation, shortness of breath, and pallor, together with hemorrhages from the mucous membranes. Pain in the splenic area not infrequently ushers in the attack. Priapism has been cited by Edes and others as an early symptom of the disease. The abdomen soon becomes noticeably increased in size, due to the enlargement of the spleen and various lymphatic structures. There is considerable fluctuation in the size of the spleen; examination may show the organ to be hypertrophied to such an extent as to reach the spines of the ilia, while a subsequent examination, after a lapse of some hours, may reveal the organ decreased to one-half of its former size. The involvement of the lymphatic glands, especially those of the neck, is very common. These glands are of stony hardness, and apt to produce pressure symptoms, impeding respiration. Vertigo is constant, and is generally caused by the intense anemia. The skin is of a pale, ashen color; sometimes it has a dirty yellow hue, but, as in chlorosis, the patient may preserve the healthy hue of the cheeks, which appearance is very deceptive. Various skin-lesions, as noted in the pathology, are apt to develop, and subcutaneous edema is rarely absent. The pulse is quick and compressible; hemic murmurs are

heard at the base of the heart and in the vessels of the neck. The circulatory disturbances are simply those of intense anemia. The liver is nearly always enlarged, and gastro-intestinal symptoms prevail. Diarrhea and vomiting are occasionally met with. The tendency to hemorrhage is a marked feature of the disease, and cases are recorded in which the loss of blood in a single flow has been such as to endanger the life of the patient. Nervous symptoms, as a rule, are not well marked; of these, headache and melancholy are the most constant.

**Diagnosis.**—The diagnosis of leukocythemia is often surrounded with many difficulties. Hodgkin's disease and the condition of leukocytosis are apt to be confounded with it. In Hodgkin's disease we have either no increase of the leukocytes or, if any, only a very moderate one, and if there be an increase, it is in the neutrophiles. Again, if leukocytosis occurs, it is liable to occur late in the disease. Between this and leukocythemia it is only during a remission that the diagnosis could be questioned. Leukocytosis can be differentiated by a failure to find myelocytes, an increase of neutrophiles and lymphocytes, and also by the absence of pathologic red cells and by the number and proportion of the neutrophilic leukocytes. Typhoid fever can easily be differentiated by the absence of leukocytosis. Frequent counts of the red and white cells and microscopic examination of stained slides are the only methods of making an absolute diagnosis.

**Prognosis.**—The course of the disease is usually chronic, and the prognosis is extremely grave.

**Treatment.**—Cases have recovered. Absolute rest is of primary importance. The personal hygiene should be carefully attended to; easily digested diet should be given, attention being directed to the quantity of food given at each meal. Arsenic has proved by far the most valuable of all drugs recommended in the treatment of this disease. Fowler's solution in increasing doses is perhaps the most efficient method of administering the drug. Quinin, iron, and strychnin prove valuable only as general tonics. Symptoms should be treated as they arise. The operation of splenectomy has proved disastrous; whereas in Banti's Disease the removal of the enlarged spleen offers the only hope. Stengel and Pancoast have used X-ray therapy in a manner that promises much. In successive settings the rays are thrown upon the epiphyses of the long bones. Their initial report was a remarkably favorable one and the treatment certainly merits an extended trial. Heretofore this treatment had been applied to the spleen, with indifferent results.

### **Anemia, Pseudoleukemia Infantum (Von Jaksch)**

This disease, first described by Italian investigators as splenic anemia and (in 1889) by Von Jaksch under the above designation, is associated with an enlargement of the spleen and a moderately enlarged liver, sometimes enlargement of the lymphatic glands, with a decrease in the hemoglobin and red blood-cells and a degree of leukocytosis. Nucleated red blood-cells have been described, and poikilocytosis may occur. It is a disease that occurs in early infancy and usually runs a favorable course. The child is very pale and waxen in appearance. Some recent writers consider it a secondary condition and think that the changes noted depend upon some gastro-intestinal condition and the age of the patient. Monti and others have found a large proportion of its subjects markedly rachitic. Other children have shown unmistakable evidence of congenital lues. We are inclined to agree with certain German authorities (Jopha, etc.) who consider these cases as but unduly severe anemias occurring at the end of the lactation period.

### **Progressive Pernicious Anemia**

**Definition.**—A grave progressive form of anemia, dependent upon hemolytic disturbances, characterized by the presence of abnormalities and a great reduction in the number of red corpuscles without a corresponding loss of hemoglobin, and almost invariably ending in death. Rotch, however, reported a recovery in a baby. Cabot studied the blood in this case and verified the diagnosis.

**Etiology.**—The true cause of this disease is unknown. It has been asserted that ptomains absorbed from the alimentary canal were instrumental in its etiology. This, as well as the infectious theory, has not been substantiated. The disease is regarded as a destruction of the blood, and not as a disease of the blood-making organs. The destruction theory is upheld by the deposit of pigment through the various organs and the presence of jaundice and hemoglobinuria. Pernicious anemia occurs in middle life, but occasionally may be seen in children. It is, however, rare in the earlier years of life.

**Morbid Anatomy.**—The skin presents a pallid, yellowish cast, and this pallor may be shared by the mucous membranes. The muscles in contrast to the external organs are remarkably red in color. A considerable amount of serous effusion is occasionally met with, especially in the pleura and pericardial sacs. Patches of ecchymosis are frequently observed upon the skin, the mucous and serous membranes, and upon

various organs of the body. Small hemorrhagic extravasations are frequently met with upon the retina in the neighborhood of the optic disc. Fatty degeneration of the heart is most constant. The appearance of the endocardium is particularly striking; it is caused by degeneration, and from the peculiar mottled appearance has been called "tabby mottling." The lungs, as well as the spleen, present no constant pathologic lesions. The liver is not infrequently enlarged, pale, and shows evidences of fatty degeneration. Many observers have attached special significance to the condition of fatty degeneration and hypoplasia of the connective tissues found in the stomach, but this condition may generally be looked upon as a result, rather than a cause, of the disease. The pancreas and kidneys are usually softer and larger than normal and show evidences of fatty degeneration. Marked degeneration has been seen in the posterior columns of the spinal cord, and the direct cerebellar tract (Burr), the cervical region being markedly affected. The changes in the bone-marrow are the substitution of red bone-marrow, as in fetal bones, for fat; the presence in this marrow of a larger number of red nucleated cells and megaloblasts and also larger cells called gigantoblasts. These bone-marrow changes are secondary.

*Blood Changes.*—On puncturing the skin, the blood does not flow freely and is very thin and pale, though oftentimes of a dirty brown. On account of its thinness it is often difficult to get enough to fill the pipet preparatory to counting, and after standing for a short time the corpuscles separate from the serum. Rouleau formation may be absent. Spreading the films is unsatisfactory for the same reason. The red cells are decreased in number in this disease beyond that found in any other condition. At the first examination a count of 2,500,000 is to be expected, and a few cases have been reported in which only 360,000 red corpuscles existed. A count below 500,000 is not very unusual. An apparent recovery sometimes occurs during the course of treatment, and a case has been reported in which the count returned to normal. After such a remission the relapse rapidly throws the count down to a very low figure. Hemoglobin is reduced, though not proportionately to the red cell diminution.

The red cells show many changes both in the size and shape of the cell. In health about 12 per cent. of the red cells measure  $8\frac{5}{10}\mu$ , and in this disease we find megalocytes, or giant cells, that measure from  $8\frac{1}{2}\mu$  to  $12\mu$ , though these rarely constitute more than 12 per cent. of the red cells. The commonest size is  $10\mu$ . These are only found in health in the new-born and under pathologic conditions in the adult.

These megalocytes may be oval or pyriform. Microcytes also exist, but have no special significance. The color of the individual red cells varies a good deal. The megalocytes are often paler than the normal cells, and the microcytes may take a very deep stain, while others appear purely as shadows of cells in which we see only a faint ring or a small amount of granular matter. Some, and especially the megalocytes, take both stains (polychromatophilic), and others show small dark granular points, which condition is known as "pinctosis." Vacuoles are found in the protoplasm of these altered cells. All these phenomena are evidences of degeneration. The shape of the cells is changed very much: they may be pear-shaped, oval, star-shaped, rod-shaped, lance-shaped, fusiform, crescentic, or kidney-shaped, constituting the best-marked example of poikilocytosis. In fact, some specimens show very few normal appearing cells. Nucleation of the red cells is said to be a common feature of this disease, a fact that a few writers deny. It takes great patience and time to determine positively that this sign is absolute. Nucleation may occur in a poikilocyte, in which case it is called a "poikiloblast." It occurs in cells of all sizes, so that we find microblasts, normoblasts, and megaloblasts. The more megaloblasts that one finds, the worse is the prognosis, and especially so as regards time. Nuclei may be found free in the serum of the blood. The red cell takes a high degree of stain. Cabot puts the average number of white cells at about 4200; the only other change that occurs is a relative increase of the small lymphocytes.

**Symptoms.**—The term "progressive pernicious anemia" expresses the clinical conditions of the disease, which begins with a slight systemic disturbance, the disease progressing with more or less rapidity to a fatal termination. Increasing languor denotes the development of the affection. Oftentimes there is an early rise of temperature, with marked irregularities. Shortness of breath and vertigo are constantly complained of, and there is a great tendency to attacks of syncope. Toward the end extreme weakness confines the patient to bed. The skin presents a lemon-yellow color (apricot hue) and is harsh and dry. Palpitation of the heart is always present and becomes extremely marked upon the slightest exertion. Endocardial murmurs are rarely absent, and the apex-beat is usually displaced considerably. The heart-sounds, although strong at first, rapidly become weak and muffled, showing evidence of fatty degeneration in the heart muscle. The pulse is short and weak, and a venous murmur is readily elicited in the vessels of the neck. Patients affected with pernicious anemia are, as a rule, flabby, but reasonably well rounded. The bodily strength rapidly

decreases, showing marked muscular degeneration. Patches of ecchymoses are often observed on the skin and mucous membranes, and edema of the extremities is most constant. The appetite is soon lost, and digestive disturbances are often present. As the disease progresses hemorrhages frequently occur without seeming provocation, and blindness, due to extravasations near the optic disc, has often been observed. There are no nervous symptoms peculiar to this disease, those present resulting from the aggravated condition of anemia.

**Diagnosis.**—A careful history must be elicited in a suspected case of the pernicious forms of anemia in order to exclude symptomatic anemias. The peculiar appearance of the patient and characteristic changes in the blood, as seen microscopically, together with the history of the case, are, as a rule, conclusive in making a diagnosis. It is not always, however, so easy to make a diagnosis as one would think from reading the account of the characteristic cases. The absence of emaciation, the failure of iron to produce results, the hemorrhages, the fever occasionally occurring, and the complexion of the patient should always be considered in a difficult case. Poikilocytosis is not associated with pernicious anemia alone, but is found in any other severe anemia. The presence of megaloblasts and giantoblasts is of great weight when found as favoring pernicious anemia. In chlorosis the relative amount of hemoglobin is decreased, while in pernicious anemia it is proportionately higher. In the latter the size of the corpuscles is usually larger, which is not the case in chlorosis, though nucleated red cells and poikilocytosis may exist. Rarely would secondary anemia give us any trouble. If it should, it would probably be in a case of malignant disease, in which case the points already mentioned should serve to distinguish the form of the disease present, so far as the blood is concerned.

**Prognosis.**—The prognosis in pernicious anemia is extremely unfavorable. At times there are temporary improvements, but a fatal termination is almost certain, either from the disease itself or from some intercurrent affection.

**Treatment.**—Of all medicinal agents, arsenic has proved the most beneficial in pernicious anemia and to a certain extent has even influenced the prognosis. It is generally advised that this drug be given in tablets, or at any rate alone. Good results have been obtained from the use of the chlorid of arsenic, and Fowler's solution has also been highly recommended. Combinations of iron and arsenic have sometimes been used with good effect, but the former drug is of less use in this form of anemia than in others. Stimulation of the patient is of great importance. Equalization of the blood pressure should be at-

tempted, either by the use of intravenous injections or massage. The condition of the stomach and intestines is of the greatest importance, and great care must be used in the administration of remedies that the digestive apparatus be not disturbed. The use of bone-marrow has been tried in this disease, but has not proved of direct value.

## SPLENIC ANEMIA

**Synonyms.**—ANEMIA SPLENICA; SPLENIC PSEUDO-LEUKEMIA;  
BANTI'S DISEASE

**Definition.**—Splenic anemia is a grave type of anemia, accompanied by splenomegaly, hepatic cirrhosis, edema (possibly ascites), a tendency to hemorrhage and marked asthenia. The leukocytes are not increased, however, but leucopenia is the rule.

Splenic anemia was a name conferred by H. C. Wood as early as 1871. Banti's disease, though often accorded a separate nosologic place, is classified by Anders and Boston as a terminal stage of splenic anemia.

**Etiology.**—This disease is more common in male infants, in children and in young adults than it is in females and in older people. (Wood and Fitz.) Its cause is unknown. Banti looks to some toxin produced by the enlarged spleen as the cause of the disease; but the investigations of Chiari and Marchand (Japha) suggest that inherited lues may be responsible for the enlarged and lobulated livers found in some cases.

**Pathology.**—As yet, there is much to know concerning the pathology of splenic anemia. The spleen is found much enlarged, sometimes as large as the spleen of true splenomyelogenous leukemia. The liver is usually diminished in size, and exhibits cirrhotic changes. Free fluid may be present in considerable quantity in the peritoneal cavity, and purpura and edema may be noted.

**Symptomatology and Clinical Course.**—The disease may last for years, though in young children its course is usually much more rapid. Banti states that the disease begins with enlargement of the spleen; but others state that the initial stage is one of high-grade anemia and muscular weakness. In the next stage, however, there is no doubt about the existence of splenomegaly. Pain is also present in the region of the enlarged organ. Hemorrhages from the stomach, bowel or kidney may occur, and hepatic cirrhosis, moderate ascites and edema of the lower extremities are frequent clinical features. Banti states that the patient perishes of hemorrhages under dropsical conditions.

As one might anticipate, hemic murmurs are often detected over the heart and great vessels.

The blood exhibits oligochromemia (50 per cent. hemoglobin, or less), oligocythemia of less degree (say 3,000,000 erythrocytes), leukopenia with a preponderance of lymphocytes, poikilocytes and normoblasts.

**Diagnosis.**—The absence of leukocytosis excludes the possibility of mistaking this disease for one or other of the various forms of leukemia. We have suspected leukemia, however, before Dr. Duncan made a careful examination of the blood. The investigator should carefully exclude malaria, syphilis, tuberculosis and some long-continued form of suppuration, before making a diagnosis of splenic anemia.

**Prognosis.**—Untreated by splenectomy, the disease progresses more or less rapidly toward a fatal termination. On the other hand, we personally know of four cases that have recovered after removal of the spleen.

**Treatment.**—Splenectomy should be performed by a skilled surgeon. Should the patient be profoundly anemic, or should he be suffering from the effects of a very recent hemorrhage, we should advise having the splenectomy preceded by direct transfusion of blood. Such a course had been outlined by one of us in the case of a three-year-old boy that he saw with Dr. Claggett. Unfortunately for us, and we believe for the boy, the patient drifted into other hands, and a more supine policy was pursued.

### Hodgkin's Disease

**Synonyms.**—PSEUDOLEUKEMIA; LYMPHATIC ANEMIA

Properly, this disease should not be classed with diseases of the blood. It is described here simply to differentiate it from lymphatic leukemia.

**Definition.**—Hodgkin's disease consists of a hyperplasia of the lymphatic glands, associated with anemia and occasional lymphoid growths in the liver, spleen, and other organs.

**Etiology.**—The etiology is obscure; it is a disease of early life, and is more prevalent in males than in females. Among those diseases which are supposed to bear a predisposing influence syphilis, tuberculosis, malaria, and rachitis may be mentioned.

**Pathologic Anatomy.**—There is no pathologic lesion that distinguishes Hodgkin's disease from leukocythemia save the absence of

leukocytic infiltration so constantly seen in the latter disease. The lymphatic glands in all the structures of the body are affected. In some instances one solitary gland, or different groups of glands, may show intense hypertrophy. Occasionally secondary infection supervenes and suppuration results. The spleen, liver, and kidneys are commonly enlarged, the spleen being especially so. The bone-marrow shows degenerative changes and evidences of lymphatic tissue formations are to be seen.

*Blood.*—The blood shows, in the first stage of the disease, no appreciable change unless it be a diminution of the red cells. When the case becomes markedly anemic, the blood count may fall to 2,000,000, and if the anemia should continue severe, nucleated red cells are found. The white corpuscles are not increased; they may be normal, a little under, or rarely a little over, and should any inflammatory action occur in the glands, they are, of course, increased, and the multinucleated cells predominate. The hemoglobin is decreased out of proportion to the decrease of the red cells.

**Symptoms.**—A slowly developing anemia is the attending phenomenon marking the progress of Hodgkin's disease. The patient complains of headache from the onset; vertigo and palpitation are rarely absent, and a gradual increase of weakness marks the progress of the malady. The symptom of chief importance clinically is the enlargement of the lymphatic glands and of the spleen. As before stated, any group of lymphatics may be affected, but the submaxillary and cervical glands are most commonly involved. In many instances hypertrophy of the glands on one side of the neck, followed by the involvement of those on the other side, usually marks the beginning of an attack. Hemorrhages are common, and small patches of ecchymosis are scattered here and there over the entire surface of the body. There is usually slight fever, but it is decidedly irregular and is inclined to be paroxysmal. At times there is considerable edema beneath the eyes and around the ankles, bearing evidence of cardiac insufficiency. The bowels, as a rule, are constipated, and although the stomach is at first retentive, nausea and vomiting may later occur.

**Diagnosis.**—Distinction must be made between pseudoleukemic and enlarged tuberculous glands. The negative results of blood examination and a tendency of the gland to soften and break down will point to the presence of tubercular disease. Much difficulty is often experienced in distinguishing malignant tumors (lymphosarcoma) from the tumefied glands of Hodgkin's Disease. The diagnosis cannot always be made, but in many cases the recognition of the slower

and more irregular growth of the tumor and the involvement of the superficial tissues will point toward malignant disease. In such cases, it is certainly a justifiable procedure to remove a portion of the gland tissue for microscopic examination.

**Prognosis.**—The chances for the arrest of pseudoleukemia are exceedingly meager, and the outlook is most unfavorable.

**Treatment.**—Here, again, as in the treatment of pernicious anemia, arsenic has been found to do the most good. Hygienic treatment should never be neglected, and much benefit is often effected by the removal of the patient to new surroundings. Iron, phosphorus, cod-liver oil, and strychnin have been used with benefit in the treatment of the disease. Preparations of the glandular substances have a valuable place here: Phosphoalbumin, bone-marrow in fluid form, or tabloids. These last should be gradually increased to do the most good. Local applications to the affected glands may be practised, and in many cases do good, temporarily at least. Surgical interference by the removal of the diseased glands, although allowable in the early stages of the disease, has thus far given negative results. The Roentgen ray has also been used and oftentimes with temporary beneficial results. The patients usually relapse, however.

## SECONDARY ANEMIA

For purposes of classification this term is applied to those conditions of blood poverty not cytogenic in origin.

**Etiology.**—Holt gives a very complete list of the causes of simple or symptomatic anemia. He cites delicate parents, ancestral tuberculosis or tuberculosis in the child, syphilis, hemorrhage, suppuration, chronic effusions, diarrhea, malignant disease, mineral and other poisonings, the toxins of the infectious diseases, improper feeding, unduly delayed lactation, bad hygiene, etc. The school life so often seems responsible for the anemia that some of the German authorities actually give it a separate place (school-anemia).

**Pathology.**—These cases are not often fatal, so we shall restrict ourselves to a description of the blood changes:

The red cells are diminished in severe cases and may fall to a very low figure. A low blood-count, however, is very rare. The cells may be paler than normal, may contain nucleated cells (normoblasts and, very rarely, megaloblasts), or may be reduced in size. The hemoglobin is decreased, but not constantly. Sometimes its decrease is more marked than the decrease of the red corpuscles would indicate.

The leukocytes are usually increased in number; they may be normal and very rarely decreased, depending upon the condition to which this anemia is secondary. When there is a leukocytosis, it is due to an increase in the neutrophiles in most cases.

In malignant disease a very severe leukocytosis is found at times. Sarcoma in childhood is an exception, the average leukocytosis in this disease being about 16,000. The red cells show no changes except late in the disease. After any hemorrhage there is a change in the blood depending upon the amount of blood lost and the character of the case. Soon after the hemorrhage has taken place and a drop of blood is withdrawn, the number of red blood-corpuscles in the individual specimen does not show a material decrease, for at this time there is only a diminution in the quantity of the blood, but as soon as serum has been withdrawn from the tissues to attempt to make up the blood volume, we find a diminution of the red cells to a cubic millimeter on account of the dilution of the blood. The hemoglobin, too, decreases. Changes in the red cells are a diminution in size, color, occasionally poikilocytosis, and a nucleation of the red cells. There is marked increase of white cells, the neutrophiles predominating. The blood presents no characteristic changes, if any, in hemorrhagic diseases; in hemophilia the blood withstands the effects of hemorrhages better than in other conditions. In scurvy there is a diminution of red corpuscles and a decrease of hemoglobin out of proportion to the decrease in the red cells, and a moderate increase of leukocytes. In advanced cases there may be nucleated red cells, polychromatophilia, and poikilocytosis.

**Symptoms.**—The characteristic pallor of the skin and mucous membranes will usually give evidence of the patient's general condition. Cabot tells us, however, that we may so be misled. He trusts only the results of the blood examination. The pulse is soft, full, and rapid. Hemic murmurs are often present, and may be heard in the vessels of the neck and at the base of the heart. The heart is frequently enlarged, later becoming dilated. Palpitation is constantly present and in cases of even moderate severity. Pulsation of the cervical vessels is often seen. Edema of the feet, especially at the latter end of the day or at night, may occur. The skin is harsh and dry, but at times perspiration has been observed. In severe cases febrile paroxysms and ecchymoses occasionally arise, and attacks of syncope give evidence of the deficiencies in the cerebral circulation. Very frequently the hands and feet are cold; vertigo and ringing in the ears may be present as evidences of marked circulatory disturbance. The digestion

is weak and the appetite capricious. In some cases there may occur respiratory changes, the breathing becoming hard. A resonant dry cough constitutes a troublesome feature. Among the nervous symptoms of this condition headache, disturbed sleep, and neuralgic pain are those most commonly complained of. Emaciation is more or less marked, and the patient seems or feels depressed. There is a general lack of vitality, this finding expression in the whole appearance of the patient.

**Prognosis.**—The prognosis depends upon the cause. Symptomatic anemia is generally amenable to treatment, save in cases of tuberculosis and neoplasm. When syncope is frequent and prolonged, the outlook is not so promising.

**Diagnosis.**—It is often difficult to separate symptomatic from essential anemia. A careful inquiry into the patient's general history and a thorough microscopic examination of the blood will aid us in this differentiation.

**Treatment.**—The original source of the anemia must be sought for and treated. In children the regulation of the diet and care of the intestinal tract often work miracles, for upon these most anemias depend. The anemia following hemorrhage requires prompt and energetic measures. Cardiac stimulants, such as ammonia, strychnin, alcohol, and camphor, must be used. To maintain the blood pressure where the depletion has been great, intravenous or subdermal injections or rectal irrigations are invaluable. For intravenous injections Osler recommends the use of the following:

Distilled water.....	1000 parts
Sodium chlorid.....	5 parts
Sodium hydroxid.....	1 part
Sodium sulphate.....	25 parts.

This should be given to the patient in a horizontal position, the head being low and the limbs bandaged. Other diseases must be treated according to the various indications. For the existing anemia a soluble form of iron should be given internally and arsenic may be used in combination with advantage. Above all, open air, sunlight, and a good supply of digestible food are essential in any plan of treatment.

**Tubercular Diseases.**—In tubercular disease there is no increase in the white cells. On the other hand, the lymphocytes are increased (Lymphocytosis). A leukocytosis appears when cavity formation occurs in the lungs, but not with a simple tubercular deposit. So long as tubercular glands and tubercular deposits are simple—that is, there

is no mixed infection—no leukocytosis is found, but when the infections become mixed, it will at once appear. Cases of tubercular meningitis have of late been reported in which leukocytosis has been found, though it is possible that some other element may have entered into the condition. In one of McKee's cases, the lumbar puncture fluid revealed tubercle bacilli and the staphylococcus pyogenus aureus. Rotch reports an increase of white cells in a child dying of miliary tuberculosis. The red cells are affected according to the degree of anemia.

*Inherited syphilis* presents a diminution of red cells and a marked leukocytosis. *Rickets* or *rachitis* presents the same condition.

*The Blood as a Guide in the Diagnosis of Appendicitis.*—The examination of the blood is of vast importance in the diagnosis as well as in the treatment of appendicitis. Following the well-known relationship of infection, pus-formation, and leukocytosis, the accompanying facts will be found of practical value. In simple catarrhal appendicitis the increase of the white cells is slight. Should there be an increasing leukocytosis during the course of the disease, it would be safe to assume that the germs had penetrated into the body of the organ, and that the peritoneal side of the appendix was becoming involved, and pus was forming, demanding operative procedures. With perforation of the appendix and in the fulminating type there is marked leukocytosis.

The blood count is useful as an indication for treatment, and the presence of a leukocytosis with pain and tenderness in the right iliac fossa and rigid abdominal muscles should always demand an immediate operation, and a case showing no leukocytosis could more safely be postponed until after the acute stage might pass. Watching it carefully, however, by daily blood counts, would keep us safely posted. One word of warning must be said, however: Do not place dependence upon the hemotologic investigation alone. It is most valuable in connection with the physical finds.

*Blood in Pneumonia.*—In pneumonia the blood presents a large amount of fibrin. There are no special changes in the red blood-corpuscles, nor is there a very pronounced anemia after the disease has run its course. The leukocytosis of pneumonia has been very carefully studied, and the following facts ascertained: if the infection be mild and the patient strong, it is slight; if severe and the patient reacts faintly, there is no leukocytosis and the result is fatal; with a severe infection and a vigorous reaction the leukocytosis is very marked and the prognosis is guardedly favorable.

The following table, originally prepared by Caspar Sharpless, will assist in the differentiation of diseases:

Disease	Leukocytosis	Lymphocytes	Neutrophils (Polymorphonuclear)	Red cells	Hemoglobin
Typhoid fever.....	Absent (Leucopenia)	Relatively increased	Decreased	Decreased	Proportionately decreased
Typhoid with complications.	Present	.....	Increased	Decreased	Proportionately decreased
Scarlet fever.....	Present	Decreased	Increased	Decreased	Proportionately decreased
Measles.....	Absent (often leucopenia)	.....	.....	No change	No change
Smallpox.....	Marked on third day	.....	Increased	Much decreased	Proportionately decreased
Erysipelas.....	Marked	.....	Increased	Decreased	Proportionately decreased
Diphtheria.....	Marked	Rarely increased	Increased	Marked early decreased	Proportionately decreased
Influenza.....	Leucopenia	.....	.....	No change	No change
Typhus fever.....	Leucopenia	.....	.....	No change	No change
Follicular tonsillitis.	Moderate	.....	.....	No change	.....
Acute rheumatism	Moderate	.....	Increased	Markedly decreased	Markedly de- creased
Septicemia.....	Marked	.....	Increased	Markedly decreased	Proportionately decreased
Abscess.....	Marked	.....	Increased	Decreased	Proportionately decreased
Meningitis.....	Marked	.....	Increased	Slightly de- creased	Proportionately decreased
Peritonitis.....	Marked	.....	Increased	Slightly de- creased	Proportionately decreased
Pericarditis.....	Marked	.....	Increased	Slightly de- creased	Proportionately decreased
Pleurisy.....	Marked	.....	Increased	Slightly de- creased	Proportionately decreased
Malaria.....	Absent.....	Relatively increased	Decreased	Decreased	Proportionately decreased
Pneumonia.....	Marked	Decreased	Increased	Decreased	Proportionately decreased
Pertussis.....	Present	Much in- creased	Actually in- creased but relatively decreased.	.....	.....
Tuberculosis.....	Absent unless mixed infec- tion	Increased	Decreased	Decreased	Decreased
Rickets.....	Moderate	Increased	Decreased	Variation in size	Decreased

In pneumonia there is a decrease of the eosinophiles and in scarlet fever an increase.

### THE HEMORRHAGIC DISEASES

Hemorrhages from mucous membranes, or into cutaneous or other tissues, may be but symptomatic of a great many morbid states, some

of which have been considered in previous sections of this work. Thus, in the hemorrhagic diseases of the new-born, bleeding dominates the clinical picture, and in most cases, the cause of it is probably infectious in nature. Syphilis, too, is a cause of hemorrhage in the new-born, bleeding from the umbilicus, and epistaxis being especially common in hemorrhagic disease of luetic origin. It is claimed that blood-vessel change is here at fault. Again, in the consideration of infantile scurvy, we have found the whole pathology and symptomatology practically dependent upon hemorrhage. In this disease, the cause appears to be a nutritive or metabolic one. Biliary pigments in the blood are often responsible for hemorrhages, as we have seen in the study of malignant jaundice in early life. Toxins, of bacterial origin, are also potent factors in the production of hemorrhages, as we shall see in the study of the various infectious diseases of childhood. Bacteria themselves have been found in purpuric and other hemorrhagic foci (*Bacillus* of Letzerich, streptococci, staphylococci, etc.). Various atrophic and cachectic states may be responsible for hemorrhages. Thus we have seen the dire significance of abdominal purpura, or the wasting incident to chronic ileo-colitis; and in leukemia and Banti's disease, hemorrhages have been mentioned among the causes of death. Lastly, a tendency to hemorrhage may be inherited, and then the diminished coagulability of the blood appears to be at fault.

It is hoped that these preliminary remarks enable the student to take a broad and comprehensive view of the whole subject of hemorrhage in early life. It is also intended that he shall perceive some of the inherent difficulties of the subject, not the least of which may be diagnostic.

Many able workers have toiled in this morbid domain: Immermann, who believed that the total quantity of blood was too great; Henoeh, who contributed to our knowledge of the purpuras; Grandidier, who insisted upon the diminished coagulability of the blood, and who wrote illuminatingly on hemophilia. We have mentioned the names of several authorities who have placed our knowledge of infantile scurvy upon its present firm basis. More modern investigators include Letzerich, Babes, Finkelstein, Osler, Legg, Sahli, Zuppinger and Hecker—all adding important material or verifying the results of previous investigators. But despite this mass of able work, there yet remains much to be learned concerning these hemorrhagic affections.

Practically, we may begin by making an important division of the whole subject: 1. Hemophilia, a disease in which there exists a hereditary and persistent tendency to bleed. 2. The purpuric disease, in

which the tendency to hemorrhage is more or less transient—lasting for greater or less periods of time.

### HEMOPHILIA

**Synonyms.**—BLEEDER'S DISEASE; THE HEMORRHAGIC DIATHESIS

**Definition.**—Hemophilia is usually a congenital disease, in which the subject shows a persistent tendency to spontaneous or traumatic hemorrhage. It has been called "the most hereditary of diseases." It is far more common in males, and is usually transmitted through the female side of the family.

**Etiology.**—It has just been stated that the disease is most often an hereditary one, and that it most commonly descends to successive generations along the maternal side of the house. As a rule, the female transmitters of the disease remain free from the tendency to bleed. Such a hemorrhagic tendency has been traced through a family tree for more than 200 years, and Osler learned that bleeders had appeared in the seventh generation of the same family. Male children are affected much more often than females: according to Grandidier, 13 times as often; according to Rachford, 112 times as often; Legg, 11 times as often; Stemper, 4 times, and Ettinger, 3 1/2 times as often. The condition is very rarely observed in the new-born; though some of the hemorrhages of very early life are probably dependent upon it. The tendency usually appears before the end of infancy, however, in 65 per cent. of the cases before the end of the second year. If the subject escapes until the tenth year of life, he may usually be considered safe. The disease is more common in temperate zones than in other latitudes, and Germany contributes more cases than any other country in the world; England and the United States being close seconds. When hemorrhages are present the coagulation time of the blood may be normal or slow. During the intervals between bleedings, the coagulation time is usually very slow (Sahli, quoted by Hecker). Wright has found the coagulation time retarded 30 or 40 minutes or more.

It is our firm impression that a number of the unexplained hemorrhages of infancy and childhood are probably dependent upon this disease. Two very recent cases, within our experience, have served to accentuate this impression. The first patient, a boy baby of 18 months, was seen with Dr. Hermann. The maternal grandmother had suffered a number of severe hemorrhages following dental procedures and other slight trauma. The mother had also shown a

tendency to bleed unduly upon slight provocation. At the time he was seen by one of us, he had suffered, for two days, a number of hemorrhages from the bowel. He was startlingly pale; but aside from this, and other evidences of severe anemia, the physical finds were absolutely negative. He had had hemorrhages from the bowel on two previous occasions. He has suffered from them on one occasion since. During the last period of hemorrhage, at our suggestion, he was seen by Dr. Dorrance. Dr. Dorrance found his coagulation time normal, and prepared some human blood serum for injection. In another patient, a girl baby seen with Dr. Charles McCreight, there had been severe hematemesis. In her childhood, the mother of the patient had had an attack of epistaxis, in which the three attending physicians had despaired of her life. This baby, like the first, presented no objective finds other than the remarkable bleached appearance.

Again, there may be no obtainable family history. A number of years ago, one of us was called upon to look after a splendid baby boy, because his sister had bled to death. The sister was seven years old, when she had an adenectomy and tonsillectomy performed in a western city. The operation was not followed by severe hemorrhage, but continuous oozing of blood continued for three days and finally induced a fatal termination. The bleeding was uncontrollable and the attending physicians viewed her case as one of hemophilia. The second baby never showed any tendency to bleed.

**Pathology.**—The anatomic finds may be practically nil. The blood will probably show delayed coagulation and the evidences of severe secondary anemia. Blood may be found in the stomach or intestines; in joints or under the skin (purpura or hematomata). Evidences of secondary inflammation may be found in the affected joints. Gangrene occasionally accompanies severe cutaneous lesions. Gingivitis, catarrhal conditions of the nose and throat, catarrhal or ulcerative lesions in the gastro-intestinal tract, unhealed circumcision wounds, etc., may reveal the origins of the hemorrhages. In many cases, however, no such points of local trauma or disease are discoverable. Vascular changes have been observed microscopically in a few instances—fatty degeneration of the intima and enlarged endothelial cells, with swollen nuclei.

**Symptomatology and Clinical Course.**—The hemorrhages are usually designated traumatic or spontaneous. It is probable that there is always some slight trauma, even when the evidence of such is lacking. Thus the two babies whose cases were just cited, had sharp abdominal pain immediately preceding their hemorrhages. One had diarrhea.

Is it not probable that some disturbance of digestion or slight inflammatory lesion induced the bleeding?

Hemorrhage from the nose or throat may occur as a result of catarrhal processes or operative trauma; from the gums as a result of caries, gingivitis or dental procedure; from the penis as a result of sacro-dotal or other circumcision; into the skin or subcutaneous tissue as a prompt sequence of slight external injuries, etc. One of our cases, seen through the courtesy of Dr. Geo. B. Wood, finally perished from persistent epistaxis. A brother had died as a result of the rite of circumcision.

Spontaneous hemorrhages, so called, may be preceded by lassitude, vertigo, sensations of cold (Hecker) or pain. Such symptoms might readily accompany the digestive or infectious process that served to induce the hemorrhage.

The hemorrhages are characterized by their uncontrollable persistence rather than by mere severity or size. As previously stated, they may take place from the gums, nose and throat, stomach, bowel, penis, and other mucous surfaces. They may be cutaneous or subcutaneous, when the lesions are likely to be fewer in number and greater in size than in the purpuric diseases.

Hemorrhages into joints are fairly common accompaniments of this disease. These joint hemorrhages may occasion no subjective symptom other than pain, and no objective one other than limitation of motion; but severe secondary inflammations may ensue, and then the patient develops fever, and the local signs of inflammation become pronounced. Ankylosis may eventually result. Gangrene is a rare result of extensive cutaneous or subcutaneous hemorrhage.

The blood changes have been noted.

Hemophilia is a dangerous disease, 60 per cent. of the patients perishing before the eighth year of life (Hecker). The total mortality is 87 per cent. Rachford says that less than 15 per cent. of the patients survive. The subjects become marvellously "bleached out" after every hemorrhage; but they usually survive one or many periods of bleeding, only to perish finally from that cause.

**Diagnosis.**—A careful elicitation of the family history may at once reveal the probable nature of the affection, or at least, may prove suggestive.

Aside from the striking pallor of the patient (resembling that of a patient late in chronic parenchymatous nephritis), the severe secondary anemia, and the delayed coagulation time, there is a remarkable paucity of clinical finds. To our minds, this absence of systemic

symptoms, such as one would find in purpura hemorrhagica; of enlarged spleen, such as one would detect in leukemia of Banti's disease, etc., is in itself very suggestive. If subcutaneous hemorrhages are present, they are usually less numerous than the lesions of purpura; while on the other hand, the individual hemorrhages are more massive. Hemorrhages into joints may usually be distinguished from peliosis rheumatica, in which the lesion is periarticular, or from Henoch's purpura in which we have abdominal pain of great severity. In this differential decision, the X-ray may prove of great service (Hecker).

**Prognosis.**—The extreme gravity of the affection has been considered. We have also called attention to the fact that most patients survive a number of hemorrhages, only to die eventually from one that is too large or that proves uncontrollable.

**Treatment.**—Most authorities lay much stress upon prophylaxis, and rightly so. Grandidier states that the women of the affected families should be persuaded not to marry. But human nature is human nature, and the woman, herself healthy, who listens to such advice must indeed be a veritable Minerva. The child that inherits the bleeding tendency, should theoretically dwell in a tropical or sub-tropical country. When at all feasible, this should be insisted upon. Rachford thinks that the relative freedom from catarrhal affections in these warm latitudes is responsible for the relative immunity enjoyed by the dweller there. Throughout his childhood, the subject of the diathesis, should be protected from injuries, from disease of the mouth, teeth and gums; from catarrhal processes; from digestive disturbances, and from dental or other operative procedures. The diet should always include fruit, particularly sub-acid fruit, in abundance. If perforce, operations must be performed, we would earnestly advise the medical attendant to study the coagulation time of the patient's blood. If this is found retarded, human blood serum should be injected intravenously or subcutaneously; or human blood may be withdrawn and immediately injected into the patient. Direct transfusion, of course, brings to mind the questionability of any operative procedure. The internal administration of thyroid gland (dissicated) may appreciably hasten the coagulation time, and this is another method that should always be tried out. From an early period the patient should be taught how hazardous certain occupations may prove for him; and his education should be shaped to prepare him for a calling free from physical menace.

Treatment of the hemorrhages: Instead of giving serotherapy last place as is so frequently done, we shall give it first. In view of much

recent work in the treatment of the hemorrhagic diseases of the newborn, we should also prefer human serum to animal serum, 10 to 20 c.c. may be injected or a similar amount of human blood may be freshly drawn from the median cephalic vein of the donor, and immediately injected into the subcutaneous tissue of the bleeding child. The father, if healthy, makes an ideal donor. In an emergency animal serum may be withdrawn; but it usually takes longer to get it than it does to secure human blood. If the measure proves of service, we may advise its repetition through the childhood of the patient.

A number of other measures may prove useful. In the main, gelatin has disappointed us; though we continue to give it internally in hematemesis and bleeding from the bowel. Despite the adverse finds of Robertson and Illmann concerning the influence of the calcium salts upon coagulation time, we continue to find these salts of service in the treatment of hemorrhage. We have seen prompt action in too many cases to believe the results merely fortuitous. We have employed the lactate of calcium, giving it in mucilage of acacia, and using from 5 to 15 grains 3 times a day. They are given for a few days only. The time-honored tincture of the chloride of iron, we have used with benefit in a number of cases of bleeding from the stomach. It is apparently a valuable styptic in these cases, and its use as a hematinic is continued after the cessation of the bleeding. We give large doses at first, and give them often. Later it is given in smaller amounts, and much less frequently. Again, the use of thyroid gland may prove a life-saving measure. If the bleeding part can be reached, the local administration of some epinephrine solution (1-1000) may promptly stop the hemorrhage. Monsel's solution is another valuable local styptic. In intestinal hemorrhage, Hecker advises the rectal injection of lead or aluminum acetate in 1 per cent. solution.

### THE PURPURIC DISEASE

**Synonyms.**—PURPURA; THE PURPLES; BLÜTFLECKENKRANKHEIT

**Definition.**—Van Harlingen defines purpura as an affection of the skin characterized by the appearance of hemorrhagic spots of various sizes, and accompanied or not by the occurrence of similar hemorrhages in the mucous membranes and viscera.

Small hemorrhagic areas, from flea-bite like lesions up to the size of a pea are designated petechiæ. Streak-like areas of hemorrhage are styled vibices, and large lesions are called ecchymoses. Allen (Prince Morrow's System) says that all of the lesions ordinarily included in systematic treatises dealing with diseases of the skin, none are less

entitled to a place than the various purpuras. From the etiologic and pathologic standpoints, we deem it wise to include them in the diseases of the circulation. Purpuras may be seen side by side with urticaria, and the exudative erythemas and are probably closely related in origin to these affections.

The **etiologies** of the various purpuric affections will be considered under their respective headings; but in general, it may be said that they are caused by circulating toxins or circulating bacteria themselves, and that these poisons act deleteriously upon the vaso-motor system, the blood-vessels or the blood.

**Pathology.**—"The blood escapes into the tissues through a ruptured vessel or by diapedesis, or there may be a mere transudation of coloring matter. The process is situated in the papillæ and the upper layers of the corium, whence the blood may pass up between the cells of the epidermis and to the deeper tissues, and about or into the hair follicles and sweat glands" (Hektoen & Riesman). Fatty degeneration and hyaline change have been observed in the blood-vessels; but such cases are few and the changes may have been due to some long-standing disease like tuberculosis or syphilis. Stasis probably plays a rôle in some of the erythemas accompanied by purpura. Hemorrhage may also take place into bullous lesions (*Purpura bullosæ*). Emboli containing bacteria, or actually composed of bacteria have also been found in the hemorrhagic area (Letzerich, Finkelstein, etc.). The lesions occurring in mucous membranes may be similar to the cutaneous spots, or free hemorrhages may take place from them. Hemorrhages may also occur in organs, joints or other tissues. Cutaneous and mucous membrane lesions may terminate in ulceration or even ingangrene.

The purpuras observed in early life may be classified conveniently under six headings: 1. Symptomatic purpura; 2. purpura simplex; 3. purpura fulminans; 4. purpura hemorrhagica; 5. purpura rheumatica; 6. Henoch's purpura.

### 1. SYMPTOMATIC PURPURA

The following tabulation of the principal causes of symptomatic purpura should prove of service to the student, even though it may be open to some scientific objections.

**Drugs:** The iodides, mercury, quinine, antipyrin, salicylic acid and the salicylates, chloral, chloroform, ergot, potassium chlorate, benzoic acid, phosphorus and the mineral acids.

**Snake bites and bites or stings of insects:** Rattlesnake, copperhead or moccasin bites, cobra venom, spider and flea bites, etc.

**Infectious diseases:** Small pox, diphtheria, scarlet fever, measles, epidemic meningitis, pneumonia, septicemia, septic endocarditis, tonsillitis, tuberculosis, malaria, gangrenous stomatitis.

**In infancy:** Hemorrhagic disease of the new-born (often septic), hereditary syphilis, jaundice, scurvy, secondary anemias, ileo-colitis (probably infectious and usually terminal, atrophy (terminal) and leukemia.

Nervous causes do not play the same parts in childhood that Weir Mitchell and others have shown that they do in the production of the purpuras of adults. In the latter, as we know, they may accompany neuroses or organic nervous diseases.

## 2. PURPURA SIMPLEX

This is the mildest form of purpura, being accompanied by no constitutional disturbances or by systemic manifestations of an almost negligible character.

**Etiology.**—Of this nothing definite is known. Van Harlingen states that it sometimes occurs in malarial patients. Occasionally the subjects have dwelt in damp houses or under other unhygienic conditions; but the disease may occur in previously healthy children, and in the best walks of life. It is probable that intestinal toxins are sometimes responsible.

The known pathology has been discussed at sufficient length.

**Symptomatology and Clinical Course.**—The lesions may appear without prodromes. On the other hand, the patient may have experienced preliminary anorexia, lassitude, headache and vertigo; or either constipation or diarrhea may have been present. There is no fever; at most only a slight subfebrile range of temperature. The lesions are small, varying in size from a flea bite to a lentil; though rarely larger blotches appear. Still more rarely, actual bullæ may form, and hemorrhages take place into them. The individual spot is dusky red at first; changes quickly to purple, and then passes more slowly through various shades of brown and yellow. It usually takes about ten days to disappear completely. At its height, the individual lesion does not disappear on pressure. The disease is sometimes prolonged almost indefinitely by the successive appearance of individual lesions or crops of lesions. The spots first appear upon the lower extremities and usually show a predilection for extensor surfaces. The arms may be affected later, but as a rule the trunk and face are least affected or wholly spared. Similar spots may be observed upon mucous surfaces;

but free bleeding from the mucous membranes does not occur. The course of the disease is usually from two weeks to a month; but if the patient is permitted to walk about, the affection may continue almost indefinitely (Van Harlingen).

**Diagnosis.**—This is relatively easy. The absence of systemic disturbance and the characters and situations of the purpuric blotches lead one to a correct conclusion. The spots are most likely to be confounded with flea bites; but in the latter, central punctures may be observed. The absence of the known causes of the symptomatic purpuras usually exclude them from further consideration.

**Prognosis.**—This is good; though even mild purpuras should be viewed with some concern, as the type of the disease may change and assume a more serious character.

**Treatment.**—The patient should be kept at rest in bed as long as the tendency to cutaneous hemorrhage remains active. The room should be cool and well ventilated. The diet should be light and nutritious, and should include the vegetable acids (citric, succinic, etc.). Milk, eggs and green vegetables probably represent the best foods; while lemons, limes and oranges furnish the desired acids. Warm astringent baths (oak-leaves, etc.) are advocated by high authorities. The mineral acids, particularly aromatic, sulphuric acid, are time-honored remedies. If the malarial element can be proved or even if it is strongly suspected, quinine should be given. In intractable cases calcium lactate should be tried for three or four days.

### 3. PURPURA FULMINANS

**Synonyms.**—FULMINANT OR FOUDROYANT PURPURA

**Definition.**—This is a severe form of purpura in which the skin lesions are very large (Ecchymotic). It may be accompanied by hemorrhages from mucous membranes, but often death takes place in from 12 to 48 hours, and in some cases it may occur before mucous membrane lesions have occurred.

**Etiology.**—There is much evidence to support the French view that this form of purpura is often but a malignant (foudroyant) type of one of the infectious diseases. Thus Rachford cites a case in which the patient died in 12 hours. This patient presented no clinical evidences of diphtheria, but another child in the same room had diphtheria at the time. We have seen small pox take this fatal purpuric form in a 12-year-old girl at the "St. Christopher's Hospital for Children."

**Symptomatology.**—The disease is characterized by the sudden appearance of large purpuric patches. These are soon attended by marked systemic disturbances, such as low muttering delirium and other evidences of the so-called typhoid state. There may be high fever, or the temperature may be normal or even subnormal. Hevre (quoted by Van Harlingen) reported a case in which death ensued in 10 hours.

**Diagnosis.**—This may be aided by the knowledge of some existing epidemic or by the presence of some infectious disease in the same household. The extreme severity of this type of purpura readily distinguishes it from all other forms.

**Prognosis.**—This is absolutely bad and no known treatment has proved of avail.

#### 4. PURPURA HEMORRHAGICA

**Synonyms.**—MORBUS MACULOSIS WERLHOFFII; HEMORRHAGIC PURPURA

Werlhof wrote upon the purpuras in the early part of the eighteenth century, and since that time, the Germans have been accustomed to apply his name to this disease; but according to Van Harlingen, there was nothing in his description to entitle him to this distinction.

**Definition.**—Purpura hemorrhagica is an infectious disease, in which the purpuric lesions are accompanied by hemorrhages from mucous surfaces, and possibly into other tissues.

**Etiology.**—The disease is almost certainly infectious, various micro-organisms (see above) having been recovered from the blood or from the lesions themselves.

**Symptomatology.**—The disease is more common in girls and in the undernourished. According to some, it is more common at puberty. After several days of such prodromal symptoms as fever, chilliness, anorexia, vertigo and diarrhea, purpuric lesions appear in various localities. These are usually more extensive than those of purpura simplex. Hemorrhages may also occur from the nose, mouth, throat, stomach, bowel, lungs and kidney. More rarely, cerebral or meningeal hemorrhage may ensue. The latter will be evidenced by the occurrence of hemiplegia. Anemia of a severe grade may result very rapidly, as Osler and others have shown. This presents the characteristics of a severe secondary anemia. Palpitation, vertigo, dyspnea and headache may result from the anemia. The temperature usually ranges between 100 and 103; but in severe cases it may reach 104 or 105 (Anders and

Boston). Much difference of opinion seems to obtain, concerning the fatality of this disease, but most cases recover, probably three-fourths of them. The usual course varies between several weeks and several months. When death ensues, it is usually in the cases with high fever and large hemorrhages, or else in the cases that tend to assume a more or less chronic form.

**Diagnosis.**—Usually this is not difficult. The constitutional symptoms, the purpura and the hemorrhages from various membranes, are the most important features to consider in arriving at an opinion. The age of the patient, the absence of a familial tendency to bleed, the freedom from former hemorrhages and the presence of fever, exclude hemophilia. The absence of joint pains and the exudative erythemas, serve to distinguish it from peliosis rheumatica, and the absence of these same manifestations and of abdominal crises, separate it from Henoch's purpura.

**Prognosis.**—It has been remarked that the outlook is grave in the cases marked by high temperature and large hemorrhages, and in those that run protracted courses. Usually, the outlook is guardedly favorable.

**Treatment.**—Rest in bed in a cool, well-ventilated apartment, and a bland nutritious diet are again necessary. Peptonized milk, egg orangeade or lemonade and gelatin should be the sheet anchors of the diet when hemorrhages are occurring from the stomach or bowel. Turpentine is a drug of signal value in these cases. A favorite palatable prescription of ours is:

R.	Oil of cloves.....	℥ vi
	Oil of peppermint.....	℥ xxiv
	French oil of turpentine.....	℥ ij-ijj
	Glycerine .....	fl. ℥ ss
	Mucilage of acacia.....	q. s. ad ft. fl. ℥ iij
	Mix.	

SIG.—℥ j three times a day, after eating.

Once more, we feel obliged to commend the employment of calcium salts in full dosage for three or four days. When epinephrine solutions can be locally applied, they may stanch severe hemorrhages from mucous membranes.

Iron, particularly in the form of the tincture of the chloride or of Monsel's solution is of value as a styptic and a hematinic.

In a very severe case, we should employ the use of human serum or human blood, and this failing, we should resort to transfusion.

Laxatives should not be used, but the lower bowel should be emptied

by lavage with warm salt solution. The drop method of proctolysis may well be employed to atone for less of bodily fluid.

## 5. PURPURA RHEUMATICA

**Synonyms.**—RHEUMATIC PURPURA; ARTHRITIC PURPURA; PELIOSIS RHEUMATICA; SCHONLEIN'S DISEASE

**Definition.**—This is a form of purpura accompanied by exudative erythemata, painful joints, and fever of a moderate grade.

**Etiology.**—Concerning the relation of this disease to rheumatism, there exists much difference of opinion. Baginsky states that it bears no relation to the rheumatic disorders. Hecker calls attention to the periartritic rather than synovial nature of the process. Among American advocates of the non-rheumatic view Rachford may be cited. Most of these opponents of the rheumatic hypothesis follow Schonlein, in stating that the heart and pericardium are spared by this disease. On the other hand, a most formidable list of authorities regard the rheumatic nature of most cases of arthritic purpura as wellnigh indisputable. Some state that it cannot always be proved. Mracek of Vienna, all of the noted English authorities who have done so much to add to our knowledge of rheumatism (Cheadle, Payne and Poynton, etc.) and Osler and Holt in this country, are exponents of the rheumatic origin of this disease. We have seen arthritic purpura, accompanied by endocardial and pericardial disease a number of times. One child appeared at the Woman's Medical College Hospital with swollen ankles, erythema nodosum and peliosis. She had an enormous heart, with ample evidence of pericardial adhesions. On the other hand we have seen the affection without involvement of the heart. We think it highly probable that several infective agents, or toxic agents may cause such symptoms; just as a number of them may cause urticaria, etc.

Mild cases of arthritic purpura are seen in childhood; but more cases of it are usually seen between 20 and 30 years of age. Osler records a case seen with Musser with endo-pericarditis. Is it not probable that these age differences simply furnish us with additional evidence concerning the clinical mildness of joint rheumatism in childhood. Unfortunately, however, this mildness means far from a promise of immunity to the pericardium and the heart.

**Pathology.**—Only two points need accentuation; the periarticular nature of the lesions and the association of the purpura with urticaria and erythema nodosum. Hektoen and Riesman describe the disease as "associated regularly with the rheumatic disorders."

**Symptomatology.**—The disease often begins with sore throat and fever. Usually, the temperature is not very high; but it may reach 102 or 103. Polyarthritis follows, though, according to Hecker, the X-ray reveals periarticular hemorrhages. Purpura appears in the region of the affected joints and in various portions of the body—usually upon the legs at first. In most cases, this is associated with urticarial wheals (*purpura urticans*) and erythema nodosum. These lesions may or may not be most marked over affected joints; though most frequently they are. There may be marked edema of the involved lower extremities. Albuminuria may occur, but not fatal nephritis, as in Henoch's purpura. Osler has twice observed sloughing of a portion of the uvula. Ulcerations of the affected skin areas have also occurred. Most cases recover though the disease may prove resistant to ordinary rheumatic measures. "Relapses may occur, and the disease may return at the same time for several years in succession" (Osler).

**Diagnosis.**—The existence of mild fever, polyarthritis, purpura and urticaria, etc., furnish sufficient evidence for a diagnosis of Schonlein's disease. A history of an initial sore-throat makes the diagnosis more sure. The absence of abdominal pain distinguished the disease from Henoch's purpura.

**Prognosis.**—So far as recovery from the attack is concerned this is good; but we must array ourselves with those who assert that the patient is often left with a damaged heart.

**Treatment.**—The patient should be in bed, clad in night-drawers or a pajama suit of wool. The room should be sunny and well ventilated. The diet should be of the same type as that mentioned in the treatment of simple purpura. In our experience, the salicylates have proved of service in relieving the joint pains, if they have not served to influence the purpura and the erythema. Strontium salicylate and aspirin are the preparations that we usually employ. A child of five years may receive 3 grains every three hours; a child of ten years, 5 grains. A method suggested by Dr. Wm. E. Robertson, we can indorse heartily in the treatment of the rheumatic affections of childhood. The patient is given three successive doses of Dover's powder, acet-phenetidin and acetyl-salicylic acid (aspirin). The third dose is followed by a hot-air bath or an electric sweat. It is our custom to give the three doses at two-hour intervals. Robertson claims that the induction of this free sweat transforms an otherwise weakening process into something useful. It serves to rid the economy of a large amount of toxin. The affected joints are covered with:

R. Guaiacol.....	q.s. ad ft. 5 per cent. ointment
Ichthyol.....	q.s. ad ft. 10 per cent. ointment
Iodide of lead.....	25 grs.
Lanoline.....	1 3

The joints are then swathed in oiled silk, covered by cotton batting and bandaged.

## 6. HENOC'S PURPURA

### Synonym.—ABDOMINAL PURPURA

**Definition.**—This is a form of purpura peculiar to childhood, accompanied by slight fever, joint pains, violent intestinal colic, vomiting and bloody stools.

Henoch observed four cases in 1868, and reported them in 1874. In a splendid monograph, Osler reported eighteen cases in his contribution to the *Jacobi Festschrift*.

**Etiology.**—The cause of this affection is not known. Some speak of it as Schonlein's disease plus abdominal pain, diarrhea and bloody stools. These cases are observed in childhood, however, while the classic peliosis rheumatica occurs in young adults. Koplik thinks that the affection may be due to some intestinal toxin, and the marked abdominal symptoms favor that view.

**Pathology.**—One of the patients included in Henoch's original report died of nephritis, and Osler reports a case of hemorrhagic nephritis (fatal), and one case in which a nephritis developed and became chronic. Other than this we know no more of the pathology of this disease than we do of the structural changes in the other purpuras.

**Symptomatology and Clinical Course.**—A patient seen by one of us in consultation with Dr. Wm. Evans of Philadelphia, presented this disease in so typical a form, that we shall record his case at some length.

The patient was a three-year-old boy.

**Family History.**—The mother of the boy had an attack of purpura rheumatica one year ago. She was under Dr. Evan's care, and made a prompt recovery when salicylates were administered in full dosage. There is nothing else of importance.

**Personal History.**—The child was breast fed in infancy, and has received an antiscorbutic diet since that time. Barring an attack of tonsillitis some months ago, he has been very healthy. The present illness has lasted for 16 days. He was seized with vomiting, fever and slight diarrhea. The following day he developed a rash that ex-

hibited macules, large urticarial lesions and petechiæ. His initial temperature was 103; but since then it has ranged between 99 and 100. He soon developed a slight swelling of the left knee.

He was treated as his mother had been, at first with apparent success. In six days, however, the eruption reappeared upon the legs. This consisted of petechiæ and large areas of angio-neurotic edema. At a later period, the latter lesions appeared upon the face and abdomen. For ten days he has suffered from violent attacks of abdominal pain, from vomiting and abdominal distention. The only abdominal tenderness detected has been over the areas of angioneurotic edema. Nor has there been any rectus spasm. For the last few days, purpuric blotches have appeared upon the palms and soles. Last night he had a great deal of pain, and lost much sleep in consequence. He has been unable to retain anything on his stomach during the last 24 hours.

**Status Presens** (Oct. 1, 1907).—The patient is well nourished, and does not impress one as a very sick child. His eyes appear normal. He is a good nasal breather. The tongue is quite clean, though it is pale and tooth-marked. His tonsils are not large. There is no discoverable adenopathy; indeed, the cervical nodes are hard to detect. The heart and lungs appear normal, and the chest is well developed. His pulse rate is 100. The liver is enlarged, its lower border being detected 1 inch below the costal margin. The spleen is felt far back, and also extends an inch below the costal border. No rectus spasm is detected, though he had several attacks of severe pain during the examination. His abdomen is slightly distended and tympanitic.

**The Skin.**—He still has a large urticarial lesion under the right eye. On the left aspect of the neck are some new petechial lesions, which do not disappear on pressure. The palms of the hands and soles of the feet exhibit fading purpuric blotches and a subcuticular mottling such as one often sees in scarlet fever. Over the dorsum of each foot considerable edema still obtains. Fading petechial lesions are noted over the extensor surfaces of both legs, and also over the skin of the abdomen. His urine has remained free from albumin (Dr. Evans). No examination of the blood has been made.

**The Following Treatment was Outlined.**—1. Absolute rest. 2. A diet of peptonized milk, fresh fruit juices. Vichy for drink. 3. Bismuth salicylate in 2-grain doses to be followed by larger doses of strontium salicylate later on. 4. Atropine sulphate, hypodermatically, in 1/600-grain doses for abdominal pain. 5. Local applications of ice to the abdomen. 6. A daily enteroclysis of warm normal saline

solution. 7. Calcium lactate in 5-grain doses if the purpura remains obstinate. 7. Arsenic and iron during convalescence.

Oct. 3, 1907. Dr. Evans reports that the boy is feeling much better. He feels that the atropine and ice have done much to relieve the abdominal pain. Another symptom of Henoch's purpura appeared yesterday, when the little patient had several stools containing blood. The boy is bright, has no elevation of temperature and is very hungry.

Oct. 10, 1907. Dr. Evans reports that the boy is well on the road to former health.

Henoch's purpura evidences a tendency to recur at intervals of weeks, months or even years.

**Diagnosis.**—The purpura, erythema, joint pains, moderate fever, colic, vomiting and bloody stools readily furnish the medical attendant with sufficient material for a constructive diagnosis. This is indeed a definite symptoms-complex.

**Prognosis.**—In the main, this is good, but the urine should be carefully watched lest a nephritis develop insidiously. Nephritis may result fatally.

**Treatment.**—The treatment outlined in the description of Dr. Evan's case amply illustrates our views upon this subject. Just before the patient mentioned was seen by one of us, "Hecker and Trumpp's" valuable little Atlas had come into his hands. It was Hecker's suggestion concerning atropine that led to what we consider a brilliant result.

## CHAPTER XIII

### DISEASES OF THE RESPIRATORY ORGANS

#### DISORDERS OF THE UPPER RESPIRATORY TRACT

Diseases of the upper respiratory tract are common in children, and their early treatment is especially important, for when neglected, disastrous effects follow upon the health and development of the child.

Inasmuch as the treatment of many of these affections is largely surgical, it requires a special knowledge and training on the part of the laryngologist. Therefore description of the various operations and instruments would be out of place here. It will be our endeavor, however, to give a brief description of the principal affections, with general suggestions for their treatment.

**Etiology and Pathology of Diseases of the Upper Respiratory Organs.**—In a brief article on nasal diseases of infancy and childhood it is well to confine ourselves to generalities, and to mention merely the more important causes which produce the pathologic changes in the upper respiratory tract in children, and the immediate consequences of these pathologic changes in their effect on the growth of the nasal organs, producing changes of development in the whole system of the child.

Among the chief causes of nasal disease, and particularly of nasal obstruction, are infection from any source, want of cleanliness in early life, and climatic and temperature influences, causing the ordinary acute coryza of infancy. From whatever cause, the first result is a swelling of the mucous tissues and a primary diminution in the amount of the secretion. Later, however, the amount of the secretion is much increased, though it is ropy, tenacious and usually mucopurulent in character. This obstructive pathologic change necessarily interferes not only with the proper respiration, but also with nutrition of the infant, inasmuch as suckling from either nipple or bottle is impossible when the nose is obstructed. If nothing is done toward reducing the swelling and inflammation within the nasal cavities, a state of subacute and, later, of chronic inflammation and tumefaction of the turbinates ensues, the normal development of the nasal organs is interfered with, and deformities result.

In order to understand this more thoroughly we must remember

that in the new-born the vertical division (septum) between the anterior nasal cavities is entirely composed of cartilage, which gradually, as the child grows older, is replaced in its posterior portion by the perpendicular plate of the ethmoid. This in its descent pushes the anterior cartilaginous plate forward, and thus produces the well-marked outline of the nose in later childhood and adolescence, which in infancy is so ill defined. Again, the posterior border of the vomer is quite oblique in infancy, but comes to occupy a vertical position with the downward development of the maxilla. The turbinate bones are developed from baso-plates. There are usually three of these turbinates; but sometimes four, five or even six.

Together with the growth of the septum and the formation of the turbinated bones the other bones of the skull and face enlarge, and it is easy to see that, as they are joined each to the other in so intricate a fashion, any lack of growth or interference with development of one bone must necessarily exert a distorting influence upon the others. It is thus that want of development of the bones forming the nasal cavities will cause a want of expansion of the dental arch, preventing space for the incisors, causing the jaw to assume a plowshare shape and the teeth often to overlap. The existence of rickets is often responsible for the accentuation of this deformity. Such want of development of the dental plates of the superior maxillary bones is always observed when nasal obstruction has been present in infancy. But it is not only the development of the face which is interfered with by obstructive nasal disease: the child is deprived not only of its nourishment, but, equally important, of its means of sustenance—namely, sufficient oxygen in the act of respiration. This deficiency of oxygen is an indirect result of nasal obstruction. It might be supposed that the child, although a mouth-breather, would be able to take in a sufficient supply of oxygen for all demands of the system; but this is not so. The nose is the true organ of respiration, because it is supplied with the necessary apparatus for warming, filtering, and moistening the air before it enters the larynx, trachea, and lungs. When, therefore, the nose being obstructed the child breathes through the mouth, the cold, dusty, and dry air impinges on the membrane of the larynx, causing inflammation and oftentimes a hacking cough. When the air reaches the smaller bronchioles, the irritation causes spasmodic contraction of the smaller bronchi and air vesicles of the lungs, so that only a portion of the lung tissue is inflated, and comparatively little oxygen is supplied to the system. This is especially noticeable in the apices of the lungs, and it is there we look for disease of the lungs in its first stages.

But long before the lungs become diseased by the inadequate preparation of the inspired air, due to nasal obstruction, the more proximate portions of the respiratory tract exhibit disturbance. If we review the diseases of the upper respiratory tract in infancy and childhood and endeavor to trace the etiology of each one of them, we shall be compelled to ascribe the ultimate cause of each to a greater or less degree of nasal obstruction or complete stenosis. By stenosis we mean complete occlusion of the nares to both inspiration and expiration, while by obstruction is meant a narrowing of these channels, one or both, which does allow a small current of air to pass. Complete stenosis is, however, but rarely met with in infancy and childhood, and is then caused either by neoplasms filling the nasal or postnasal chambers or by a congenital malformation of the nasal bones, most frequently observed as obliteration of the posterior nares by a thin slate of bone projecting across their posterior orifices (atresia). One of us saw such a case quite recently, having the opportunity to observe the baby from the time of birth until her eventual relief by two drilling operations. Partial stenosis or obstruction of the nasal chambers is, on the other hand, quite frequently met with in infancy and childhood, and is due to a variety of causes. First, and most frequently observed, is the obstruction caused by temporary and, later, permanent swelling of the tissues covering the lower turbinated bones. This tumefaction of tissue may be caused by specific infection from the mother during delivery, or by exposure of the child to extreme changes of temperature, producing what is commonly called a cold in the head or the "snuffles." In addition to the swelling, a thickening of the normal watery secretion of the organ results from the congestion, and, by its retention in the form of a thick, tenacious flake, adds considerably to the obstruction. It must also be remembered that the tissue underlying the mucous membrane covering the turbinates is composed of a network of blood-vessels which becomes distended and enormously enlarged by the stimulus of an inflammatory process in the immediate neighborhood. If this inflammatory process is not speedily checked and the membrane and secretions restored to their normal state, we soon have a chronic catarrhal condition and, in consequence, more or less permanent obstruction to respiration. A less frequent cause of nasal obstruction in children is deviation of the nasal septum, which may be due to traumatism (falls or blows upon the nose), or it may be caused by the pressure of a hypertrophy of the lower turbinated body pushing the cartilage toward the opposite nasal chamber. It must be said, however, that there are few perfectly straight nasal septa. Slight deviations are noted even in the lower animals.

Still another cause of impaired nasal respiration is the introduction of foreign bodies into the nose. They are usually introduced by the child itself, unknown to the parents, and give rise to symptoms of cold in the head, with copious mucopurulent discharge, which may be tinged with blood. Later the symptoms are those of hypertrophic catarrh, with the addition of an offensive discharge. The ordinary mucoid polypi which produce nasal obstruction are seldom, if ever, met with in infancy, but are not uncommon in later childhood, and, by the increasing pressure during their growth, produce in time noticeable deformity by a flattening and spreading of the upper portion of the back of the nose.

More important, however, than all the other causes of nasal obstruction, is the presence of hyperplastic adenoid tissue in the nasopharynx. It is said the naso-pharyngeal space in the new-born is but 3 mm. in depth, and 10 mm. in height. One may readily perceive how such a small space may be completely occluded by a moderate tumefaction of the Luschka tonsil.

Mouth-breathing is injurious, as we have said, because the inspired air is not cleansed, moistened, or warmed as it is in normal nasal breathing, and the result is irritation of the mucous membrane of the pharynx, larynx, trachea, and bronchi, which is readily lighted up into acute inflammation by cold or other systemic disturbance. Mouth-breathing kept up for a long period of time results in chronic inflammation of the mucous membrane of the respiratory tract, favoring in children the formation of laryngeal neoplasms. More serious yet is the fact that the concomitant inflammation of the upper air-passages in the exanthematous diseases is enormously aggravated by the nasal obstruction, while the irritation of the bronchioles by the dry and dust-laden air prevents its entrance into the lungs in sufficient quantities for the requirements of the system, and thus not only does the whole economy suffer, but the foundation of lung disease is often thus laid in early childhood. On the other hand, it not infrequently happens in scrofulous children that a specific or nonspecific acute rhinitis, instead of resulting in permanent hypertrophy of the turbinated tissues, is followed by an atrophy of not only the mucous membrane, but also of the turbinated bones, and the serous as well as mucous glands embedded in the membrane. Such a condition interferes in the same degree with normal respiration, and produces pernicious effects upon the whole respiratory tract and the system at large, as does nasal obstruction. The nasal chambers become too large from the shrinking of the tissues, so that the air current cannot be properly warmed in its passage, and, the glands be-

coming atrophied, the air cannot be properly moistened and filtered, so that we have the same conditions as in mouth-breathing. But to all this must be added the formation of large adhesive scabs in the nasal chambers, which, by their bulk, cause obstruction; by their presence as foreign bodies, irritation; and by their adhesiveness, denude the membrane of its epithelium and cause hemorrhage at their expulsion; and by their putrefaction give rise to the well-known and offensive odor of ozena. The latter fact increases the danger from this kind of nasal catarrh to the system at large, because of the volatile products of putrefaction which are carried into the lungs during respiration.

Nor should we close this brief chapter without a reference to the important accessory cavities of the nose, the frontal, maxillary, sphenoidal and ethmoidal sinuses. We quite agree with Makuen that the Eustachian tube and middle ear should be included in this list, for anatomically and pathologically they too are but closely related to the nasal chambers. The sinuses proper do not exist at birth and are not fully developed until the pubescent period.

### ACUTE RHINITIS

**Synonyms.**—ACUTE CORYZA; THE “COMMON COLD”

**Definition.**—Acute coryza is an inflammation of the nasal mucosa of a contagious character.

**Etiology.**—It is not a specific disease, but rather a symptom-complex that may accompany a number of the infectious diseases, or that may result from the invasion of the nares by a number of different organisms. Chief among these latter are the influenza bacillus, the micrococcus catarrhalis, the pneumococcus, the bacillus lanceolatus (Friedländer), staphylococci, and streptococci. In most of the late autumn, winter and early spring “colds,” the influenza bacillus is present in the nasal secretions. Epidemicity has been a proved fact, since 1889, when the disease began in Bokhara, and spread along the lines of commerce until it became a veritable pandemic. Infants and young children are peculiarly susceptible; though coryza is no respecter of persons and attacks most people who are exposed. Tissues of lessened resistance in the nose and nasopharynx invite invasions of these regions (*vide supra*). Again, some children, like some adults, have peripheral vaso-motor mechanisms which render them peculiarly susceptible to draughts, changes in temperature and alterations in barometric pressure. Steam-heating, imperfect ventilation; indoor life; over-dressing indoors, with its resultant relaxation of the skin,

combine to contribute more victims and to render the sensitive still more sensitive.

One can never ignore the importance of the common cold, when one has once grasped the fact that colds cost society more than all other infectious diseases.

**Pathology.**—Probably this has been sufficiently touched upon. Primarily there is marked swelling of the nasal mucosa, and congestion of the submucosa. Later there is a watery secretion, and still later a muco-purulent secretion. Permanent lesions, such as hypertrophied turbinals and adenoids, may remain after a single severe coryza, and they are almost sure to result from repeated attacks. Again, as Makuen has so forcibly taught, the middle ear is in reality a sinus of the nasal cavity, and it may be invaded by the infectious process. Older children may have the other accessory sinuses involved.

**Symptomatology and Clinical Course.**—In infants and young children, the disease is usually ushered in by slight or moderate fever, and some obstruction to nasal breathing. On the other hand, in very young infants, we have seen dangerous depression of temperature and other evidences of overwhelming toxemia rapidly ensue. Older children experience chilly sensations, headache (particularly frontal headache) lassitude, and muscular soreness. The obstruction of the nares may be very serious in the case of infants, interfering as it does with nursing and with sleep. At first the nasal discharge is scanty, thin and irritating to both lips and anterior nares. In the course of from 24 to 48 hours, the secretion becomes muco-purulent, often of a greenish-yellow or amber hue. Herpes labialis, so often designated "cold sores" are fairly common accompaniments, probably more often when the pneumococcus and the influenza bacillus are the offending organisms.

The average course of an acute coryza is from a few days to a week or two; but some "head colds" end as such, while others show a tendency to traverse the whole respiratory tract.

Complications, by continuity, may involve the eyes, the Eustachian tube, and the middle ear. The invasion of the meninges, though fortunately far more rare, may nevertheless take place by the nasal route.

**Prognosis.**—This is usually good, though the mere mention of the above complications, should lead us to view coryza as no trivial ill.

**Treatment.**—Naturally this divides itself into prophylactic and active treatment.

One of the best preventive measures is the open-air existence. The child who spends much of his time in the open has better resisting

powers and higher blood pressure; but most of all, he is less exposed to infection from contact with the subjects of coryza. When indoors, a room temperature should rarely exceed 65° to 68°. Cool sponging in the second year of life, and cold bathing for older children, are other measures that raise systemic tone and train the surface vessels to respond promptly and efficiently to external temperature changes. The matter of clothing is a difficult one, and must be answered differently in the cases of individual children; but a safe rule is to dress the child so that he is comfortable indoors, and to depend upon externals for outdoor comfort. A perspiration-bathed skin, when the child is indoors, usually betrays over-dressing. Heavy underwear, saturated with sweat becomes a menace, not a safeguard to the child. Flannel chest protectors should not be tolerated.

Adenoids, hyperplastic tonsils and other removable tissues of lessened resistance should be dealt with summarily.

With the appearance of an acute coryza, the child should be kept at rest in a cool (65° to 68°) well ventilated apartment. His diet should be liquid and semi-liquid. Usually, a mild, initial purge (castor oil) is indicated.

The time honored drugs, atropine and camphor are of signal service. The first drug lessens secretion, and the second probably acts through the circulation and nervous system to increase the resistance of the patient. Quinine is less popular than it was some years ago; but while we do not know how it acts, we cannot escape the impression that it does good, and that it sometimes aborts the attack. Hexamethylenamin is theoretically indicated, and experience seems to justify its present-day popularity.

In general, the dosage of atropine may be safely adjusted by Young's rule; but the dose should be sufficient to produce the result, and it is a safe drug. Camphor and quinine may be given in relatively large doses as children stand them well. Hexamethylenamin is administered in 1/2- or 1-grain doses to an infant, and in 2- to 5-grain doses to children. One should remember that it may produce vesical irritation and hematuria.

Local measures are of much service, and in infants, their use may be demanded. Following several of our rhinologists we first spray with a 1/2 to 1 per cent. solution of cocaine hydrochlorate containing 1 to 2 per cent. of antipyrin. After several minutes, we use a warm normal salt solution or some alkaline solution, spraying it into the nostrils very gently. This cleansing spray is followed by a bland oil, such as the one suggested by Freeman.

℞. Menthole,  
 Camphor.....a.a. gr. i.  
 Benzoinol .....fl. ʒ i.  
 M. et solve.  
 Sig.—Drop several drops into each nostril.

To be effectual, the local treatment should be carried out every three hours, the child's resistance being controlled to avoid injury from the atomizer. (See Freeman method.)

Handkerchiefs should not be used, particularly someone's else handkerchief. Squares of cheese-cloth or bits of absorbent cotton do the work just as effectually, and after use, either may be wrapped in newspaper and subsequently burned. Thus a common source of infection and reinfection is destroyed. Children should not be permitted to blow their noses vigorously, nor to occlude one nostril when removing secretion from the other. How often one sees an adult handle his own irritated member gently, and then proceed to attend to his offspring's nose as though he were actuated by some inhuman motive. Careful local treatment of the nose, also prevents the child from using his tongue in the disgusting removal of secretion from the upper lip.

After-treatment is important. For a few days the child may not stand exposure well; but in convalescence he should be inured with expedition to an out-of-door existence. Tonics are usually indicated in this stage, and should be more frequently employed.

**Vaccine Therapy.**—We have seen a few very wonderful results from the employment of stock vaccines ("mixed influenza"); but in other cases have been disappointed in their use. Theoretically, autogenous vaccines should yield better results; but practically they may be very difficult to procure and the necessary time required for their preparation may debar their use when we need them most. We have used stock vaccines for prophylaxis, early in the disease, and in intractable cases. Our best results have been secured in the last group of cases. Our experience has been confined to children. We have hesitated to use them in infants because marked pain and tenderness at the site of the injection have usually obtained for at least a day.

#### CHRONIC RHINITIS (SIMPLE AND HYPERTROPHIC)

These are but stages of the same affection, the simple passing into the hypertrophic. Both are characterized by more or less nasal obstruction—in the former, temporary swelling or turgescence of the turbinals, shifting from one side to the other; in the latter, permanent

engorgement, due to hypertrophy of the turbinate bodies. In both the secretions are apt to be thick and abundant. The cause may be repeated acute attacks or some permanent irritation within the nose, such as spurs or deviation of the septum. The indications for treatment are removal of the secretions and the reduction of the swelling and obstruction. All projections or irregularities of the septum should be corrected, and any postnasal or faucial obstruction to nasal respiration and drainage removed. In the stage before the hypertrophy has taken place an attempt to reduce the swelling by local applications (such as the following: Iodin, 3 grains; potassium iodid, 6 grains; glycerin and water each,  $\frac{1}{2}$  ounce; menthol, 3 to 10 grains in an ounce of liquid albolin or vaselin) may be made. If this fails, the turbinates should be cauterized, preferably by one of the chemic caustics, such as chromic acid or trichloroacetic acid. In the hypertrophic stage cauterization by the galvano cautery or removal by the snare may be required. In both conditions alkaline antiseptic washes should be employed and the passages kept entirely clean.

#### PURULENT RHINITIS

This is distinctively a disease of childhood. It may be the result of direct infection or the sequel of one of the exanthemata particularly scarlet fever. The symptom is a purulent or mucopurulent discharge from both nostrils, which tends to collect in scabs around the nostrils and may become offensive. The treatment consists in cleansing washes, followed by some mild astringent, such as glycerite of tannin, half a dram to the ounce of water, zinc sulphocarbolate, five grains to the ounce of water, or alumnol, five to ten grains to the ounce; and, internally, iron and cod-liver oil.

#### ATROPHIC RHINITIS

##### **Synonym.**—OZENA

It is thought by some that atrophic rhinitis is a sequel of purulent rhinitis; by others it is ascribed to scrofula or inherited syphilis. The syphilitic form should be sharply differentiated from the non-syphilitic by the history and the concomitant signs of inherited syphilis. It is, however, a distinct affection and never the result of the hypertrophic variety. Its chief characteristics are the formation of crusts throughout the nasal cavities, generally extremely fetid, and increased size of the cavities from wasting of the turbinates and membrane. The in-

dications for treatment are cleansing, stimulation, and protection. The crusts must be thoroughly removed by spray, syringe, or cotton swab. Any of the alkaline solutions above mentioned may be used, but their antiseptic properties should be increased. Thymol, which is a good antiseptic and deodorizer in this condition, may be added to any of the cleansing washes, in the proportion of  $\frac{1}{4}$  or  $\frac{1}{2}$  of a grain to the ounce. After all the crusts have been removed some stimulating application should be made. Nitrate of silver, five to ten grains to the fluidounce, or a solution of thymol, ten grains to the ounce, has been found efficient; after this an oily substance, preferably vaselin, either plain or medicated, warmed and sprayed into the nose, to protect the surface from rapid drying. Such treatment must be carried out by the physician at least three times a week at first. In the intervals the patient can use the cleansing wash as a spray or douche at home in the morning and evening, following it with one of the liquid petroleum preparations, with oil of eucalyptus or menthol added. The treatment must be kept up continuously until the crusts cease to form in the nose, or, at least, until the patient is able to keep the nose free from crusts. The patient, however, must continue to use some cleansing wash for a long time afterward. Syphilis must receive its appropriate systemic and local treatment.

### CROUPOUS OR MEMBRANOUS RHINITIS

This is not an uncommon affection in children. It is characterized by the formation of a false membrane in the nose, which can be readily detached but rapidly reforms. Constitutional disturbance is very slight. The principal symptom is nasal stenosis. One of us has laid stress upon the importance of epistaxis as a symptom of nasal diphtheria. Bacteriologic cultures have shown streptococcus and, in some cases, the diphtheria bacillus. Clinically, the two forms are identical, unless the membrane of diphtheria extends into the posterior nares. It should be clearly understood that there are two forms of nasal diphtheria, one representing the mildest variety of that disease, though capable perforce by its very mildness of spreading infection broadcast; the other postnasal or nasopharyngeal diphtheria, probably its most virulent and dreaded variety. The duration of the disease is from two to three weeks. The treatment consists in the removal of any loose membrane, cleansing, and the insufflation of iodoform (euophen or nosophen if preferred) or calomel. Dilute lime-water has been suggested by McBride. Iron and bichlorid of mercury or calomel in small doses should

be given internally. If the Klebs-Loeffer organism is found, the child should be segregated and treated with antitoxin.

### SYPHILITIC RHINITIS

The coryza of syphilis, the common "snuffles" is more frequently noted in infants than in adults. Any obstinate nasal catarrh in an infant should suggest the possibility of syphilis. The children usually appear emaciated, and skin eruptions will generally be found. There are swelling of the membrane and hypersecretion, which may be purulent and bloody. In tertiary syphilis, which rarely appears before the fifth year, infiltrations of gummatous material, ulcerations—especially of the septum—and necrosis of bone may be found. In syphilis of the nose the treatment is very important. In infants the stenosis often prevents suckling, necessitating feeding by the spoon. Tonics are indicated such as syrup of the iodid of iron and cod-liver oil. In the early stages mercurials are all-important. They can be given by the mouth or by inunction. The nose must be kept clear by antiseptic washes. Menthol in oil (5 to 10 grains to 1 fluidounce) may be used to relieve the stenosis. In the tertiary stage destruction of tissue is rapid, and the resulting deformity may be very great. The photographs (see Syphilis) illustrate such deformities, though this patient has improved vastly under general and local treatment. Iodid of potassium should be given in increasing doses up to the limit of tolerance. Recent investigations with the *treponema pallidum*, however, must convince us that Mercury is of value in all stages of syphilis. Seiler recommends the surgical removal of the infiltrated tissues to prevent destructive ulceration. Iodoform should be used locally.

### MUCOUS POLYPI (EDEMATOUS FIBROMATA)

These do not occur in infants, but are not infrequent in older children. They grow from the upper portion of the nasal cavity, but by elongation of the pedicle may occupy any portion of the nares and extend into the nasopharynx. They produce nasal stenosis and watery discharge, greater in damp weather. They do not, as a rule, cause deformity. Headache, laryngeal spasm, and asthma are often due to their presence. Polypi may be caused by disease of the accessory sinuses or by any prolonged irritation of the nasal cavities. The growths should be thoroughly removed and any underlying disease treated.

## FIBROUS TUMORS

Nasal fibromata appear as dense white or reddish tumors. They spring from the periosteum or bone. Their favorite location is at the vault of the pharynx, whence they advance into the nasal cavities, by their growth spreading the bones apart and often producing great deformity (frog-face). They should be attacked early. Electrolysis has been successful in some cases.

## ADENOID VEGETATIONS

**Etiology.**—There is normally at the vault of the pharynx a group of lymphoid glands called the pharyngeal tonsil. Hypertrophy of this tissue is known as adenoid vegetation or hypertrophy of the pharyngeal tonsil. This condition is most frequently found in childhood, as the lymphoid tissue here, as well as that in the fauces, tends to atrophy in later life. Adenoids may be congenital, but are generally of later growth, frequently appearing after measles, scarlatina, diphtheria, whooping-cough or grip. A hereditary predisposition probably exists, as they are often found in several members of the same family. They are usually associated with hypertrophy of the faucial tonsils and the pharyngeal follicles.

**Pathology.**—One observes practically the same changes in this lymphoid tissue that may be noted in enlarged faucial tonsils. The resulting mass may be soft in consistency, or because of fibrous overgrowth (hyperplasia) may be very firm. The mass may be exceedingly small, being detected only high in the nasopharyngeal vault, or it may be so large as to fill this space and invade the fossæ of Rosenmüller. It may, again, be located particularly around the orifices of the Eustachian tubes, practically occluding them. Wood and others have found tubercle bacilli in these vegetations.

**Symptoms.**—In infants the symptoms and signs may be very few; but the following are worthy of note: repeated attacks of coryza, often with little nasal discharge; hypernoea; middle ear disease; retraction of the xiphoid; cervical adenopathy, particularly of the superficial chain of glands (the old posterior-cervical). In older children, anyone should recognize the symptoms and signs of adenoids. Possibly we can make the description most lucid by outlining their possibilities for harm: 1. Constituting as they do a tissue *minores resistentia*, they constantly expose their possessor to infections. This is true not only of coryza, but also of the specific infectious diseases of

childhood. 2. As previously mentioned they render such infectious diseases as scarlet fever, diphtheria and measles more dangerous. 3. They constantly menace the ear, either in the production of catarrhal deafness or in the more serious suppurative otitis media. 4. They constitute a portal of entry for bacteria and toxins to gain access to the lymph glands and channels of the neck. Many cases of tuberculous adenitis are traceable to them. (Fig. 74.) 5. They favor the production of physical deformities. Many of these, resulting from mouth breathing have been mentioned; but we must hasten to add to that list the all too common funnel-shaped chest ("trichter-brust"). 6. The subjects of adenoid vegetations often present stunted growth of both body and mind. No doubt impaired oxygenation accounts in some part for such retardations in development; unquestionably the deafness is of importance in the production of mental backwardness; but we believe that the full explanation of these developmental failures has yet to be advanced. 7. Among the reflex phenomena that might be described as dependent upon adenoids we would mention particularly asthma, disturbed sleep, pavor nocturnus, and enuresis. After thus reviewing these possibilities for ill, it is scarcely necessary to describe the physical finds. Suffice it to say that as the student examines such a patient, from the stupid face with its drooping lines and open mouth, downward, he will find them prolific and interesting enough. Let him complete the examination by introducing his finger behind the soft palate.



FIG. 50.—ADENITIS.  
Care of Lewis Marshall, Esq.,  
M. D., Nottingham, England.

**Diagnosis.**—This is readily made by eliciting the history and making a careful examination. There can be no mistake with care.

**Prognosis.**—This must be broad in its scope and consider carefully all the possibilities above detailed. With early and thorough operation, the prognosis becomes very good. It is rare for adenoids to recur when the operation has been thorough.

**Treatment.**—This is surgical. We have come to heartily endorse the snare-wire procedure for enlarged tonsils and the punch operation for adenoids. We believe that not a curettement but a complete surgical dissection (with the punch, etc.) is the proper procedure to pursue. We further believe that it is "a hospital operation" to

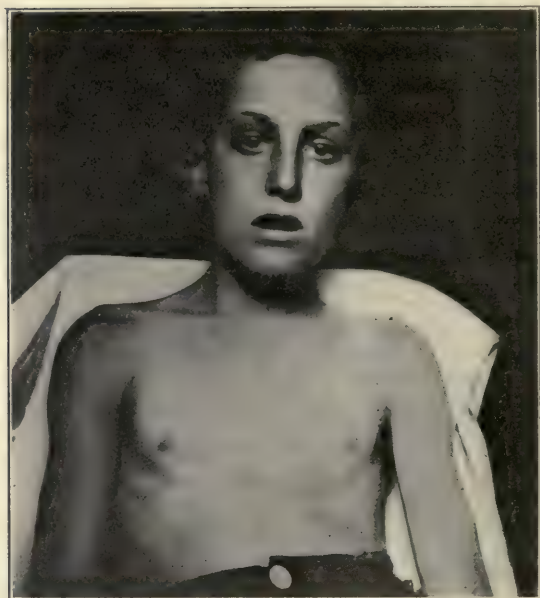


FIG. 51.—Adenoid facies with pronounced oral breathing.

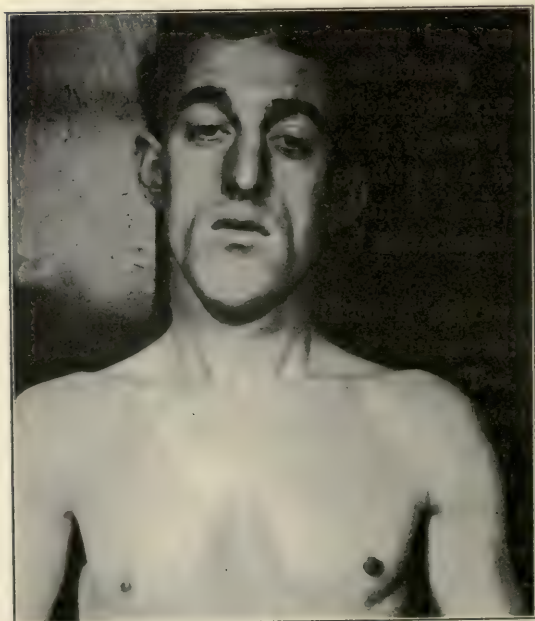


FIG. 52.—The remote results of adenoids.

be performed with all modern facilities at hand. The after-treatment is important, and not the least part of it is teaching the child to breathe properly.

In Fig. 50 are exhibited the "adenoid-facies," a vacuous expression and pronounced oral breathing.

Fig. 51 shows the remote results of adenoids in a young adult. There are noted in his case, lowered mentality, dull expression, broadened nasal bridge with narrow nares, drooping facial lines, high-arched palate, poor alignment of teeth, obtuse mandibular angle and a very acute mental angle. The chest is ill-developed; flat and depressed over the xiphoid (trichter-brust). He has incipient tuberculosis at the left apex.

### ACUTE PHARYNGITIS

This is usually caused by exposure to cold or dampness, especially in those already debilitated by hereditary influences or by living in a vitiated atmosphere. There is often a disturbed condition of the digestion along with this or acting as a cause. The attack is accompanied by fever, headache, pain in the throat, coated tongue, and constipation. Examination of the fauces may show a general redness or only streaks of congestion on the lateral walls of the pharynx and the half-arches. Hot foot-baths, aconite in small and frequent doses, laxatives, etc., constitute the general treatment. Locally, mild astringents, such as glycerite of tannin, diluted, or a single application of silver nitrate, sixty grains to the ounce, followed by mild astringent gargles, are commonly employed. Wet compresses to the neck are useful and give comfort.

### RHEUMATIC PHARYNGITIS

This is characterized by but slight congestion of the membrane, but a disproportionately severe pain in deglutition. It usually occurs in rheumatic subjects and is not frequent in childhood. Antirheumatic remedies should be given. Heat to the neck and rubbing with stimulating liniments give relief.

### RETROPHARYNGEAL ABSCESS (RETROPHARYNGEAL LYMPHADENITIS)

The lymphatic glands embedded in the posterior wall of the pharynx occasionally suppurate, forming an abscess most dangerous to life because of mechanical obstruction to the trachea, produced by pressure or edema, or by suffocation from a spontaneous opening deluging the air-passages with pus. Most of our knowledge of this

important subject has been derived from the Bokais of Buda-Pesth. It is of much importance to recognize the condition promptly, both to institute treatment, if time permits, or to meet the exigencies thus induced, and, above all, to differentiate this from simple tonsillitis, laryngeal stenosis, or impaction of a foreign body. Two varieties exist, one, the commoner, occurring in infants usually under one or two years, seldom above three; the other, rarer, resulting from caries of the cervical vertebræ, is seen only in older children.

The retropharyngeal lymph-nodes are described (Simon) as forming a chain on each side of the median line between the pharyngeal and prevertebral muscles; these undergo atrophy after the third year. The adenitis may be severe enough to produce serious local symptoms, yet stop short of suppuration; it is also occasionally associated with external cervico-adenitis.

**Etiology.**—Children are very prone to lymphatic inflammations, especially of the cervical glands. The causes of retropharyngeal abscess are usually specific infections, most often tubercular or influenzal or less frequently following scarlatina, measles, or diphtheria. The immediate cause is usually an inflammatory condition of the nasal or pharyngeal mucous membrane. Abscess sometimes occurs in children hitherto vigorous, but more readily in the weaker ones, subject to catarrhal affections.

**Symptoms.**—Abscess of the retropharynx may begin slowly or arise with alarming suddenness; indeed, sometimes death is imminent or occurs before the real trouble is suspected. The situation may be in sight on the postpharyngeal wall, or as high in the vault, or low down, where it can only be felt by the finger. We cannot impress too often or too emphatically, the importance of such digital examinations in the presence of obscure throat symptoms. The swelling may be seen nearly in the median wall of the pharynx or oftener to one side. There may have been an antecedent catarrh. There may be high temperature, loss of flesh, and other evidences of an acute suppurative process—a prostration out of all proportion to the other phenomena. The first definite symptom is usually an attack of dyspnea or asphyxia, due to pressure of the abscess on the larynx. The stridor is usually both inspiratory and expiratory. Labored mouth-breathing during sleep is usual, the head is thrown back, and there is difficulty in swallowing. The voice becomes nasal, food is regurgitated through the nose or mouth, and a squeaking cry occurs, resembling the “quacking” of a duck. The tumor sometimes shows externally. The Bokais claim that a more or less superficial lymph node is always involved at the

level of the abscess. The finger in the throat will tell most by demonstrating the position, size, and consistency of the mass.

**Prognosis.**—Death may result by suffocation, asphyxia, or drowning by pus when the disease is not recognized early enough, and rarely may come from burrowing, ulceration of the carotid, etc. If the prostration is profound, this may cause death after the pus is evacuated. The mortality is 5 per cent.

**Diagnosis.**—Instances of trouble in swallowing occurring among infants with mouth-breathing or dyspnea call for examination of the throat by touch as well as inspection; not many mistakes will then be made.

**Treatment.**—If the condition is recognized early enough, relief can be obtained by hot applications, chiefly to assist the abscess in pointing; resolution can scarcely be hoped for. When pus is evident, the cavity should be opened at once, using great care to prevent the pus from flowing into the trachea, which is best accomplished by keeping the head well forward or it may be thrown forward the instant pus is set free. It is not well to use a gag; as this alone may cause serious asphyxia. A knife is required, when the mouth should be opened and held in position with a small, narrow tongue-depressor (our device of a wire loop serves us best), and the incisions made with a short-bladed tenotome, from the side toward the median line. We cannot see the force of the claims of certain surgeons who insist on the advantages of external incision, except it may be for the cases due to Pott's disease. If the abscess is large and the tissue at the side of the pharynx is involved, and especially if there is burrowing of pus into the deeper tissue, then, in order to insure proper drainage, an incision should be made in the neck and the wound and abscess cavity packed with gauze.

### **Retropharyngeal Abscess from Caries of the Cervical Vertebra**

This variety is rare, and seldom occurs in children under three years of age. The pus-cavity is larger, forms slowly, often for months, and is accompanied by more marked constitutional depression but less sudden changes; the swelling is oftener in the median line, and not so circumscribed. The symptoms of cervical Pott's disease usually precede, though the abscess may occur before the deformity, and external swelling is more common; on digital exploration an angular prominence may be felt on the posterior wall of the pharynx. This form of abscess may open spontaneously on the outer surface below the jaw, or lower in

the neck, or the pus burrows in front of the spine; the cavity, once open by punctures or spontaneously, may refill and become a slowly discharging sinus. The treatment is incision, preferably external, and drainage. Tuberculin in small initial doses, and gradually ascending dosage, is returning to favor in the treatment of these cases. We saw a case of a boy, eight years of age, in a hospital become asphyxiated during dinner, and on thrusting the finger in the throat to extract a piece of food suspected of causing this, buried our finger in a large cavity, and a fragment of meat was withdrawn, along with several ounces of pus. Once opened, the cavity heals, as a rule, but it sometimes requires cleaning out and scraping. The cervical caries, of course, should receive its proper orthopedic treatment. A seashore residence, with life in the open air, should succeed the active treatment of the caries.

### TONSILLITIS

#### **Synonym.**—AMYGDALITIS

Tonsillitis is either acute or chronic. The acute conditions are divided into three varieties—the superficial, or catarrhal, which does not differ materially from simple pharyngitis; the follicular or lacunar; and the phlegmonous, or quinsy. Properly speaking, however, quinsy is really a peritonsillar affection. Several other forms of tonsillar inflammation are mentioned by different authorities, but practically all cases fall under these three subdivisions.

**Etiology.**—Acute tonsillitis is very common among children and is liable to occur at any age. The fact that epidemics of it occur at times points clearly to the bacteriologic origin of the disease. Certain germs have been isolated in the anginas, many of which are found normally in the mouth, and in the order of their frequency may be mentioned: the pneumococcus, diphtheria bacillus, streptococcus pyogenes, influenza bacillus, staphylococcus pyogenes, the Vincent organisms, and the tubercle bacillus. In making the cultures a mixed infection of two or more of these is not infrequently found.

A rheumatic diathesis exerts a marked influence over the anginas as a predisposing cause and the tonsil is also the principal portal of entry for the rheumatic poison. Exposures to wet and cold, also poor ventilation and unhygienic surroundings, act as exciting causes, debilitating the system, and especially the tissues of the throat, for the reception of the infections. Overexertion of the voice also prepares the soil for these germs.

**Symptoms.**—The symptomatology of tonsillitis is the same for the three varieties, varying in severity with the condition; thus we have pain in the throat, with difficult swallowing and articulation, the voice sounding as though the mouth were filled with some soft food. There is marked tenderness at the angle of the jaw on pressure, and usually a swelling is apparent. This swollen gland was formerly thought to be the posterior submaxillary; but it is really the upper gland of the deep cervical chain. The vasa efferentia of the tonsil become the vasa afferentia of this gland, so that it is well named the tonsillar lymph node. Fever always accompanies to a greater or less degree, reaching as high as  $104^{\circ}$  to  $105^{\circ}$  F. ( $40^{\circ}$  to  $40.6^{\circ}$  C.) in the severer forms, often preceded or accompanied by a decided chill. The pain often radiates to the ear.

In the *simple* form the tonsils are uniformly enlarged, injected, and bathed in thick, tenacious mucus.

In the *lacunar* form the tonsils are not always so uniformly enlarged, but one or both are markedly swollen, and dotted throughout the surface with yellow or whitish spots of various sizes; these spots are where the lacunæ or crypts are filled with débris from degenerated cells, caused by the bacterial invasion, and swollen from the occlusion of their outlets. These lacunæ or crypts discharge their contents in the latter stages of the disease, causing an offensive odor. The exudate from the crypts may extend in the severe types to the whole surface of the gland, rarely beyond, simulating diphtheria, and if such cases are seen late a diagnosis should never be made without awaiting the result of a culture.

In the *phlegmonous* form of tonsillitis, (really peri-tonsillitis) commonly known as quinsy, a more marked and general constitutional disturbance is noted, the tonsils are greatly swollen, and the pain becomes intense and throbbing. The bacterial invasion (usually of pus-cocci), probably takes place in the supratonsillar space, and the tissues surrounding the tonsil are rapidly invaded. The gland is pushed toward the median line by the peritonsillar invasion. One side is usually affected; but if both be, the tonsils may actually overlap one another and effectually close the faucial space. The tonsils themselves share in the inflammation; but the pus is not often concealed within the tonsillar substance. The lymph nodes at the angles of the jaw are almost invariably tender and swollen. Swallowing becomes almost impossible, fluids being regurgitated through the nose; the breathing is difficult and the speech much restricted; the fever runs as high as  $105^{\circ}$  F. ( $40.6^{\circ}$  C.), being irregular in its intermit-

tence, as in all forms of pus infections. The pulse may be as high as 130, and there is often marked salivation, earache, and even delirium. Headaches and backaches are common, and albuminuria is not rare.

Quinsy occurs more frequently when there are great climatic changes, usually during the cold and damp seasons. It rarely troubles very young children.

In children tonsillitis may be mistaken for the acute tonsillar and pharyngeal inflammation of scarlet fever, especially when the tonsillitis is accompanied by a rash. Tonsillitis may, however, be distinguished from scarlet fever by a history of contagion, with an onset of vomiting, a strawberry tongue, the characteristic pulse, and, finally, the peculiar rash of scarlet fever. If the patient is first seen at night-time, it is wise to withhold a diagnosis until morning. Diphtheria is claimed by some to be distinguished from follicular tonsillitis by the appearance of the false membrane, which extends to the surrounding parts, and is of a grayish, creamy tinge, curled at its edges, and, when removed, leaves a raw surface; also there may be a history of contagion and a rapid weak pulse, with marked swelling of the tonsillar lymph nodes.

No one may trust such a differentiation, but, when possible, should always proceed to make a bacteriologic examination and isolate the patient until the report comes back from the laboratory.

**Prognosis.**—The prognosis of tonsillitis is generally favorable, even in the severer cases; but one should bear in mind its possible complications and should warn the parents, so that they may cooperate more willingly in preventive measures. In quinsy, when the patient seems in greatest danger, relief is often brought by the spontaneous rupture of the abscess. It is only when ulceration takes place through the carotid or the abscess bursts during sleep, causing suffocation, that we hear of fatal results. The duration of the disease is from two days to two weeks.

**Treatment.**—The treatment consists of rest, quiet, and avoidance of exposure; the best external application is ice or cold cloths applied at least every hour or two. For pain, dry heat or poultices often give relief. Free purging with calomel or salines is of great value. In the simple form swabbing the throat with sulphate of zinc solution, twenty grains to one fluidounce, or tincture of red gum is of use. For older children gargling with extremely hot water for some ten or fifteen minutes, followed by gargles of astringents, such as potassium chlorate, two drams, or *rhus glabra*, one ounce, to a pint of hot water is very comforting and effective. As an abortive treatment we can highly recommend the use of strong nitrate of silver solutions as recom-

mended by Dr. Gleason. These must be carefully and thoroughly applied while the child is controlled in the Freeman position. No excess must be present on the applicator, for fear it might drip into the larynx. Swabbing the tonsils and throat with Lœffler's solution has in the hands of one of us proved of great benefit. The follicular form should be treated with similar gargles and astringents after thoroughly disinfecting the parts by a spray of a diluted solution of hydrogen peroxide or a direct application of the same on a pledget of cotton into the crypts of the tonsils.

We are particularly fond of the following solutions for gargles as local applications:

℞. Potass. chlorat..... 3j  
Hydrogen, dioxid,  
Glycerin,  
Aq. rosæ..... āā fl. ʒij  
M. et solve.

SIG.—As gargle or spray every three hours.

or

℞. Potass. chlorat..... 3i  
Tr. ferri chlorid,  
Glycerin,  
Aq. distillat..... āā. fl. ʒij  
M. et solve.

SIG.—As gargle or spray every three hours.

Internally sodium salicylate, two to five grains thrice daily, may at times shorten the attack and relieve the pain. Acetylsalicylic acid is another drug of great value. Calomel,  $\frac{1}{30}$  of a grain directly on the tongue, repeated half hourly, is often useful. The phlegmonous form, in addition to the above, usually needs surgical interference. Whenever pus is suspected or fluctuation felt, a free incision is instantly demanded. This is best done on the soft palate outside the line of the anterior pillar, by means of a guarded bistoury, which should be wrapped within half an inch of the point. Should pus not be found, a probe can be passed deeper with safety. Great relief immediately follows the evacuation of the pus-cavity, and the inflammation rapidly subsides. The child should always be kept in bed for a week after the subsidence of acute symptoms.

**Complications and Sequelæ.**—The complications and sequelæ of tonsillitis have only recently been recognized to be of much gravity. In the last few years many investigators have made careful studies of this subject, and it is a well-established fact that the tonsils are open gateways for the passage of many kinds of germs.

Jessen, Buschke, Boeck, Buss, Hodenpyl, Ribbert, Du Mesnil,

De Rochemont, Packard, Mayer, Morse and Osler have reported cases of grave conditions following attacks of tonsillitis. Thus it has been found that attacks of albuminuria, erythema, urticaria, purpura, rheumatism, erysipelas, orchitis, oophoritis, pleuropneumonia, strabismus, and paraplegia have been traced directly to tonsillar invasion. Phlebitis, purulent pleurisy, and even tuberculosis have followed these attacks. Endocarditis is by no means an uncommon sequela, and general pyemia has been reported as consequent upon this apparently simple disease.

Otitis media, with its mastoid and intercranial complications, has frequently been traced to attacks of tonsillar inflammation (when accompanied by adenoid growths), especially is this true following the throat troubles of scarlatina. Nor does the severity of the attack bear definite reference to the gravity of the sequelæ, which oftentimes appear late and after all the throat symptoms have cleared up.

#### CHRONIC TONSILLITIS (HYPERTROPHY OF THE TONSILS)

Chronic enlargement of the faucial tonsils may be found in infancy and early childhood, but it is much more frequent in later childhood. When present in early life, the cause is probably heredity. Later the enlargement may be due to previous attacks of acute inflammation or to bad hygienic surroundings, constitutional disease, etc. Bosworth describes two varieties of enlargement of the tonsils—the hypertrophic and the hyperplastic. In the former the glandular tissue is mainly increased, and the tonsil is rough and irregular in appearance. It is this type that is more likely to be associated with the presence of adenoid growths of the nasopharynx. In the latter the fibrous tissue is increased, the tonsil presenting a smooth and round appearance. Very often tonsils exhibit a combination of these conditions. Tonsils in which the crypts are chronically diseased and have become the seat of cheesy deposits may be only slightly or not at all enlarged, but are a source of irritation and are subject to attacks of acute inflammation. Mycotic tonsillitis, chronic in type may be observed, frequently in the tuberculous. Many symptoms are ascribed to hypertrophy of the tonsils, most of which are due to the obstruction to respiration, nasal and oral, caused by their presence. The chief symptoms are a snoring during sleep, restlessness, thick or nasal voice, liability to take cold, deafness, and tinnitus. As we have seen, however, most of these symptoms are really dependent upon the accompanying adenoids. Internal remedies have very little effect on tonsillar hypertrophy. Tonics, etc., may be given for the general condition,

which is apt to suffer in these cases. As local applications, the compound tincture of iodine, diluted; tincture of the chlorid of iron (one part to three of glycerin); glycerite of tannin, and nitrate of silver (ten to twenty grains to the ounce) may be used. Of these the iodine and the iron solutions are of the most value. They can be used in recent, soft hypertrophies. If the crypts are diseased, they may be cauterized with chromic acid, fused on a probe, or the galvanocautery point carried into the crypts. In moderate degrees of hypertrophy, also the galvanopuncture may produce shrinking and atrophy. When the tonsil is large and firm, however, excision is the only treatment to be advised; indeed we believe this to be the appropriate treatment in the vast majority of cases. The snare-wire procedure is the one that we indorse.

### DISORDERS OF SPEECH

#### **Chiefly Those Due to Anatomic Defects of the Speech Apparatus**

The importance of clear speech as a factor in mental and physical development is scarcely appreciated, and the subject fails to receive the attention it deserves. Defective speech is not always the result of defective mentality, as many seem to think, but it is quite as often the cause. The child cannot speak because it is thought to be stupid, whereas the child is often dull because it cannot speak.

The speech faculty develops not by intuition, but by imitation, and there are two ways in which this development may be retarded: first, the child's imitative capacity may be weak, while all the other faculties are strong, and then speech, which is so largely dependent upon the imitative faculty, will necessarily develop slowly and imperfectly. Again, the child may have had poor models of speech in those having it in charge, and the result is equally unfortunate. This explains why these defects are so common among the poorer classes, where the imitative faculty is oftentimes but little developed and where the speech of the attendants is careless and slovenly.

Defective hearing is also an obstruction to the normal development of speech in children. Total deafness, either before or during the formative speech period, always results in faulty speech, because the child cannot imitate what it does not accurately hear; but in addition to this often those "having ears, hear not." We have those who possess no ear for speech, just as there are those who have no "ear for music." They hear the speech as they hear the music, but the ear and brain do not make the fine distinctions so necessary for its sensitive differentiation or normal development; and so, just as the train-

ing of the ear forms an essential part in the training of the musician, and just as some ears require more training for music than others, so the training of the ear should not be overlooked in those children who are backward in the development of speech. They must be taught to hear the sounds correctly and to distinguish them, the one from the other, before they can ever learn to execute them.

Various other subjective physical conditions influence the development of speech in children. Indeed, anything which makes speech difficult or even disagreeable to the child may result in serious imperfections. Obstructions in the nostrils due to hypertrophied turbinals, irregularities of the septum, or adenoid vegetation in the vault of the pharynx act in several ways to impede speech development. They interfere with normal respiration and with the resonance of the voice; they set up a catarrhal condition of the vocal organs, and oftentimes press upon certain important nerve filaments, causing irregular choreiform movements of the muscles controlling voice and speech.

In all cases of delayed or arrested speech development these parts should be carefully and thoroughly examined and put in the best possible condition. Nasal spurs should be removed, deflected septa straightened, and hypertrophied turbinals reduced, always bearing in mind that the slightest deviation from the normal in the upper respiratory region during the formative period may render speech difficult and disagreeable, and therefore impossible, to the child who does not appreciate the importance of good speech sufficiently to make an effort to overcome even slight impediments.

We have known a small adenoid growth in the pharyngeal vault to cause stuttering of the severest type, and the defect of speech to cease immediately upon the removal of the growth. A long and curled epiglottis setting up a pharyngeal and laryngeal irritation may be the cause of the disordered speech, and we have had most excellent results, in at least one case, by the removal of its upper border.

Another fruitful source of defective speech is found in hypertrophied faucial tonsils which press upon the pillars of the palate and encroach upon the oropharyngeal resonant space. We often see, also, inflammatory adhesions binding the tonsils to the pillars, and preventing that free action of the palatopharyngeal and palatoglossal muscles which is so essential to good articulation. The adherent or imbedded tonsil should be completely removed by the snare-wire.

So important an organ is the tongue that its very name has come to be regarded as a synonym for speech, and "tongue-tie" is generally supposed to be one of the chief causes of defective speech.

By tongue-tie we mean a short frenum interfering with the movements of the tip of the tongue. This we do find, of course, in some children, and the snipping of this frenum undoubtedly gives greater freedom to the tip; but in many cases the trouble is not in the frenum alone, but in the disposition of the anterior fibers of the geniohyoglossus muscles. These fibers are too short, and they are inserted into the body of the tongue too far forward toward its tip, thus preventing some of the most important movements of this organ. And so in the majority of cases the snipping of the frenum is not enough to loosen the tongue, but an incision must be made through the mucous membrane low down in the floor of the mouth, and about one-half the width of the tongue, so that the anterior fibers of muscle are divided sufficiently far back to give the tongue its normal amount of free surface and motion. This operation has been described and illustrated by G. Hudson Macuen in the (April, 1897) "International Clinics," and is one which is more frequently indicated than the somewhat simpler method of snipping the frenum. In cases of cleft palate the operation should be done in the first year, before the formative speech period begins, one of the chief indications in this operation being to retain as much as possible of the soft palate and uvula, and to restore to them their normal functional activity. Generally in these cases there is too little velum palati after the operation and too little activity in the muscles which control it, the result being an inability to close the palatopharyngeal chink (the opening from the oropharynx into the nasopharynx), and the stream of sounding breath which should pass out through the mouth during speech is allowed to escape through the nostrils; hence all the consonant sounds except the nasals must necessarily be defective.

The most important and the most neglected part of the treatment of speech defectives is undoubtedly the training. This cannot begin too early. The child should have the best model as an example of speech, and the imitative faculty should be trained and developed. All "cute" baby talk should be discouraged, and only good forms of speech encouraged.

#### LARYNGITIS CATARRHALIS

**Synonyms.**—CATARRHAL LARYNGITIS; FALSE OR SPASMODIC CROUP; LARYNGITIS STRIDULOSA.

**Acute catarrhal laryngitis**, is an inflammatory affection of the larynx and trachea, non-contagious in nature, and excited by an acute

catarrh. The most conspicuous feature is often the "croup," or loud cry, or ringing metallic cough. It is usually followed or accompanied by tracheitis or bronchitis. It may be primary, secondary to the infectious disease, or traumatic. The lesions are found chiefly in the mucosa and lymphoid tissue of the subglottic region, and in severe cases they may be so serious as to produce laryngeal stenosis.

Laryngitis is one of the commonest of the respiratory diseases of early childhood, occurring at any time, but usually in the changeable weather of autumn and spring. It may be mild or severe, and is of importance chiefly because of the uncertainty of the diagnosis, as well as the distress that it occasions to the family and the anxiety felt by the physician, who, be he ever so skilful, not seldom fails to make a correct diagnosis on his first visit. We are called upon to distinguish, then, between false croup and true or membranous croup which is usually laryngeal diphtheria, and between false croup and laryngismus stridulus. False croup is a common malady and so is diphtheria; the other is comparatively rare. False croup occurs in isolated instances, although several members of a family may be so predisposed. Sporadic cases of diphtheria are not uncommon.

**Etiology.**—The causal factors are age, commonly between two and five years, neurotic heredity, enlarged tonsils, adenoid growth of the pharynx, exposure to cold, dampness, and disturbances of digestion. In the last few years, we have come to believe that adenoid growths play a more important predisposing rôle than is generally recognized.

**Symptoms.**—False croup comes on suddenly or may be preceded some hours or a day or two by catarrhal symptoms such as coryza and slight fever. Sometimes the larynx and trachea are involved; there is cough, but without stridor or spasm. The attack usually begins in the night, with almost no warning, except, perhaps, the cough, which changes and becomes short, deep-toned, and barking, with a peculiar resonant quality readily recognizable; the inspirations have a whistling, crowing sound; the little one exhibits surprise or terror, sits up in bed, clutches at nearby objects, especially its mother, and seems to experience some relief in holding on to objects, the way asthmatics do, which enables the ribs to become more vertical, thus assisting in securing a deeper inspiration. There may be extreme recession of the thoracic spaces and superior and inferior retraction. The cough has a metallic, hard quality, associated with dyspnea, which lasts for perhaps just a moment, or a little longer, when it lessens, and the child may, in half an hour or so, have entirely recovered and

quieted down to sleep. The attack is not likely to be repeated more than once or twice at most; but several times during the night the short barking cough may recur. Next morning the child is apparently well, with the exception of the cough, which usually remains. The attack tends to return on several successive nights. The temperature may be little above normal, or about  $101^{\circ}$  or  $102^{\circ}$  F.; some observers report a much higher temperature. Absence of fever should usually arouse our suspicions and put us on guard against a Klebs-Loeffler infection.

There is another type of catarrhal laryngitis, in which spasmodic croup does not occur at night; but in which symptoms persist during the day for several days. The "barking" cough, the whispering voice, and marked inspiratory stridor may lead one to think of membranous laryngitis. Usually, however, the temperature is higher in laryngitis catarrhalis; there is no deposit on the tonsils or pharynx, and Klebs-Loeffler bacilli are not present in smear or culture. The affection is a sthenic and not an asthenic one.

**Diagnosis.**—While it is universally recognized that membranous laryngitis may be caused by pus-cocci, yet it should always be assumed that one is dealing with a diphtheritic infection (Klebs-Loeffler) when the larynx is invaded by membrane. Practically then we differentiate between spasmodic croup (so called) and laryngeal diphtheria until *the result of the culture* is known. The following table will probably prove helpful to the student:

	Spasmodic croup	Laryngeal diphtheria	Laryngismus stridulous
The type of patient affected.	The child often has adenoid growths. It may be neurotic.	The disease may occur in any child.	It is nearly always observed in rachitic infants.
The history of exposure.	None.....	This may be obtained.	None.
Preceding symptoms.	There may have been none, or it may have been preceded by coryza or bronchitis. There may have been no antecedent catarrhal phenomena.	A history of a preceding tonsillitis, or of a suspicious nasal discharge (foculent or bloody) is often obtained. Primary laryngeal diphtheria is relatively rare.	None necessary; but digestive disturbances may have been present.
Fever.....	This is often high, though not necessarily so.	It is usually moderate and not high. It may be normal or actually subnormal.	There is no fever, unless it be dependent upon some accompanying condition.

	Spasmodic croup	Laryngeal diphtheria	Laryngismus stridulous
The pulse.....	Usually commensurate with the fever.	Often asthenic. It becomes very weak and rapid after the dyspnea has persisted for a time.	It presents nothing characteristic.
The respirations...	Noisy metallic type of inspiration, persisting for a short time, and usually at night. Retraction of the supra-sternal notch and lower chest may be present during the paroxysm.	The rate is slow and there is marked inspiratory stridor which usually comes on slowly and progressively. The air sounds as though it were being sucked through a narrow orifice. Nevertheless, paroxysms of dyspnea occur, which tend to grow more frequent and severe.	The breath is held for a while and the child seems on the verge of asphyxia. Then there is a loud crowing inspiration. This may be repeated several times before the baby can succeed in uttering a cry. It may occur at any time of day.
The cough.....	A harsh metallic bark..	It may be harsh at first, but soon acquires a characteristic whistling and stuffy character. (To anyone who has heard this cough, it is practically pathognomonic.)	None.
The voice.....	This is usually whispering, but occasionally presents the same metallic quality as the cough. The latter quality is particularly noticeable in crying.	Whispering; but in addition to this, it suggests that the expired air is being forced through a narrow chink (stuffy).	Voiceless during the attack; but a natural cry usually follows it.
Cyanosis.....	Not always present. It may be observed during the paroxysm.	A late and dangerous symptom. It is seldom observed until late in the second day of the disease. One should never await its appearance to make a diagnosis, or adopt mechanical measures for relief.	Present during the paroxysm; but disappears immediately after it.

**Treatment of Spasmodic or False Croup.**—The treatment of croup is simple but imperative. The child should be kept in one well-ventilated room, with an equable and distinctly moist temperature. The clothing should be sufficient, lest chill should occur. The bowels should be open in almost any event, calomel being the best remedy, although castor oil or salines may be used. The food had best be fluid, such as milk, guarded by alkaline water, or thin gruels or soups. For the milder varieties ipecac should be used to the point of nausea or full relaxation; it is well to combine this with soda, the powdered ipecac

being preferable, although the syrup form is satisfactory. Ipecac may be given with calomel in minute doses every fifteen minutes in a powder on the tongue, and this it is well to combine with a little soda. For a baby of a year or two,  $\frac{1}{60}$  of a grain of calomel,  $\frac{1}{20}$  of a grain of ipecac,  $\frac{1}{2}$  of a grain of soda, with a little sugar of milk, may be given dry upon the tongue every fifteen minutes. It is not wise to use severer depressants unless the fever runs high, when aconite, one-half to one drop every fifteen minutes, is of great utility, and is safe because of the ease with which its administration can be regulated. Antipyrin is useful in cases requiring nervous and arterial sedatives and when there is decided increase of mucous secretion. With the antipyrin it is well to give a few drops of brandy or some other alcoholic stimulant.

For a child two years old antipyrin,  $\frac{1}{2}$  to 1 grain, syrup of ipecac, 2 to 4 drops, sodium bicarbonate, 1 grain, brandy, 6 to 12 drops, may be given every half-hour to one hour during the severity of the attack or during the night, and at longer intervals during the following day. Pilocarpin is recommended but is dangerous, as it is too depressing to the heart; nevertheless it is sometimes of manifest value, especially in older children. Antimony is not to be used, except possibly in vigorous older children, and then with caution. There are times when opiates are distinctly useful to allay excitement or distressing cough, but they are not to be repeated too frequently, one or perhaps two doses being sufficient to relieve a violent paroxysm. A piece of belladonna plaster the size of a small coin is placed by some on each side of the throat. To relax the spasm various mechanical devices are useful. A hot poultice applied to the chest is effective. A sponge wrung out of hot water will do as well. Counterirritation to the chest with oil and turpentine or camphorated oil is useful. Local cleansing or spraying of the nose or throat is to be recommended in some cases. If the rhinitis should be severe or obstructive, albolene, containing one grain of menthol and five grains of camphor to the ounce, is both soothing and stimulating to the nares. In the case of a nervous child the bromids may aid in securing a good night's sleep; an opiate, however, is rather better, and five to fifteen drops of syrup of Dover's powder acts nicely. One of us has found ammonium iodid of vast value as a prophylactic in children who have spasmodic croup. It is also most efficient in preventing recurrences during the second night. Following the advice of the late Wm. Pepper, he has used:

R.	Ammon. bromidi .....	gr. xxxvi
	Ammon. iodidi .....	gr. xxiv
	Ammon. chlorid .....	℥j
	Syrup Tolu .....	fl. ℥ss
	Syrupi .....	q. s. ad ft. fl. ℥iij
	M. et solve.	
	SIG.—Fl. ℥j every three hours (to a child two years old).	

Kerley recommends, also, to an infant three months old tartar emetic,  $\frac{1}{100}$  of a grain; ipecac,  $\frac{1}{30}$  of a grain; antipyrin,  $\frac{1}{4}$  of a grain, every hour. In severe laryngeal spasm great relief is obtained by fumigations with calomel, vaporized on an ordinary milk warmer and alcohol lamp under a sheet tent. The Ermold lamp is better. Use ten grains in ten minutes, and let the child lie then for twenty minutes longer. This produces a copious watery secretion from the larynx.

So much importance has been claimed for the marvelous success of homeopathic treatment of croup that we asked Dr. J. Nicholas Mitchell, Professor of Obstetrics at the Hahnemann Medical College, to give us a full description of his methods which he has most kindly done. We use his own words: "For hoarse, barking, dry cough, with fever, I use a mixture of tinct. aconit. napelli (leaves), ℥j; tinct. spongiæ, ℥ij; using this, according to the age of the child, from  $\frac{1}{2}$  to 2 drops every fifteen minutes till the cough loosens. When the cough is loose and the phlegm is interfering with respiration, with occasional spasmodic, barky cough, I mix one grain of the one-tenth trituration of hepar sulphur. in from four to six teaspoonfuls of water, and give one teaspoonful every fifteen to thirty minutes. Hepar sulph. calcareum is an impure calcium sulphid." We have tried this faithfully, and with fair result.

### Membranous Croup

**Synonyms.**—TRUE CROUP; PSEUDOMEMBRANOUS LARYNGITIS; LARYNGEAL DIPHTHERIA

**Etiology.**—Membranous croup is essentially the laryngeal form of diphtheria, but is not proved to be always due to the Klebs-Loeffler bacillus. Cases that follow measles, etc., may be due to pyogenococci. The reason probably is that the culture cannot always be made from the infectious portion of the exudate. It deserves to be considered separately from the general heading of diphtheria because of its clinical features, which are those of a laryngitis. Diphtheria of the pharynx presents a somewhat different onset, features, and course. True croup may appear suddenly—with a series of phenomena oftentimes

endangering life by mechanical obstruction before the constitutional symptoms obtrude themselves at all—but as previously pointed out, the onset is usually slow. Absorption from the larynx is feeble and slower than from the pharynx; hence glandular enlargement and albuminuria are less common, nor is there the striking asthenia seen in cases of genuine toxemia from diphtheria. Postmortem, the structural degeneration in the viscera common in diphtheria is wanting, if the cultural methods and case be primarily laryngeal; but usually diphtheria of the larynx is secondary to nasal or pharyngeal diphtheria. Contagion is feeble—because the discharges from the throat and nose are less or are absent—and the course is shorter. In short, the forms of diphtheria vary so widely that there may be instances where it cannot be detected except by the secondary phenomena and complications.

**Symptoms.**—True croup differs little in its onset from false croup or catarrhal laryngitis, except that it is slower or not quite so abrupt nor so severe at first. There is the same high-pitched, ringing cough, hoarse voice, general discomfort, and quick but not weak pulse. The catarrhal phenomena are less marked or absent. Dyspnea increases slowly, and under excitement becomes profound, and alarms both parents and child. The temperature is seldom high—between 99° and 100° F., or a little over; the skin is pale and moist, and as obstruction progresses—steadily, as a rule, differing from false croup—the surface grows cyanotic. Breathing becomes much more difficult on the second and third days, and is accompanied by all the distressing features which follow this state—tossing, restlessness, irritability, etc. The child seizes on to objects to aid the respiratory action by muscular efforts.

The respiratory sounds become rough, without vesicular murmur. The symptoms, if unrelieved by treatment, progress from bad to worse, the temperature rises to 104° or 106° F., and death ensues by strangling, convulsions, or coma.

We reported a typical case in which the symptoms rapidly grew alarming, and intubation was performed by Dr. Freeman. The cough became so extreme that the junction of two ribs with the costal cartilages parted, producing hernia of the lung, which was cured by strapping. In this case the earlier bacteriologic examinations showed no Loeffler bacilli, but analysis of the material on the tube, when removed eight days after the operation of intubation, showed them to be abundant; yet at no time was there a large amount of membrane.

**Diagnosis** is to be made by careful examination, which involves exploring the throat by the finger to exclude retropharyngeal abscess

and foreign bodies. The suddenness of the onset of true croup is not so great as in false croup nor in the above-mentioned states, yet it may arise most swiftly. Bronchopneumonia has a higher temperature, as a rule, and characteristic signs in the chest. The form of dysp-



FIG. 53.—IMPROVISED CROUP TENT, MADE FROM AN UMBRELLA AND A SHEET.

nea is different too. The child is quieter; in spasmodic croup it is restless and struggles.

The **prognosis** of true croup depends on the age of the patient

and the character of the epidemic and the time in which treatment is instituted. In untreated cases the mortality is very high.

**Treatment.**—Nauseants should never be used, but antitoxin should be administered at once, in a full dose—10,000 units or more. (See Diphtheria.)

Nowhere is the serum treatment more efficacious than in these cases. Inhalation of steam is a useful adjuvant; so, especially, are calomel fumigations. The tent should be applied at once and Ermold's lamp put in operation or the practical plan suggested by Holt. This is to take an ordinary chamber-pot, and place over the top of this a strip of tin; on this is placed the calomel (ten or fifteen grains), and beneath it an alcohol lamp, with the flame in contact with the metal. Soon the white vapor of mercury rises and fills the tent; and this should be kept up from ten to fifteen minutes. (Care must be exercised not to let this be knocked over by the child and cause a conflagration.) This may be repeated every hour or two, according to the needs of the case. It affords marked relief in most cases. After the calomel has been used the tent should be removed and the room aired. Sometimes vaporized mercury causes choking, if in too concentrated a form. Salivation is rare, but may occur among the attendants, who should be warned not to put their heads under the tent. Relief is seen usually after the second or third fumigation. It should be begun as soon as the croup is diagnosticated, before dyspnea becomes marked. The operation of intubation to relieve the obstruction of false membrane in the larynx must not be delayed; certainly not until cyanosis sets in, which may only occur just before death. Tracheotomy may be performed if intubation fails to relieve. Before the use of antitoxin the mortality was 30 to 40 per cent. from these operations. Now it is much less, and decreasing steadily, partly because of the enormous help afforded by antitoxin, which is peculiarly helpful to this class of cases, and partly because the operation of intubation is more promptly and skilfully performed. This usually suffices, but occasionally tracheotomy is made to supplement the intubation when that is insufficient to afford relief.

#### LARYNGISMUS STRIDULUS

**Synonyms.**—LARYNGISMUS, LARYNGOSPASM; SPASM OF THE GLOTTIS, OR RACHITIC ASTHMA.

This is a disorder characterized by paroxysmal narrowings of the glottis, accompanied by spasmodic disturbances of respiration,

occurring and recurring at intervals. It is a neurosis of the larynx, which is otherwise healthy. It should not be confounded with false croup or with infantile asthma (laryngitis stridulosa). The paroxysm is produced by a spastic contraction of the muscles which narrow the glottis, occurs always during inspiration, and results from irritation of the recurrent laryngeal nerve or of the pneumogastric. It is asserted by some authorities to be a characteristic symptom of latent tetany.

**Etiology.**—*Laryngismus stridulus* is a disorder of infantile life, usually from the fourth to the fourteenth month, and rarely beyond the third year. The children attacked present many marks of rickets. The disease prevails in the cold months and among those too much confined indoors.

The causes are constitutional and local. It is quite well established that the constitutional disorder known as rickets is at the bottom of most, if not all, of the cases of laryngospasm, and two-thirds of the children affected present well-marked evidences of rickets. In rachitic infants nervous irritability is generally exaggerated; in them slight exciting causes—emotional, exposure, and catarrhal—readily produce morbid respiratory conditions. Hereditary predisposition is blamed by some, but this is scarcely more than can be explained by the neurotic constitution and impaired nutrition. Thiemich, Finkelstein and others believe that all the patients exhibit the electric reactions of spasmophilia. Local causes are numerous, but chiefly in the line of digestive disturbances, protracted or acute.

**Symptoms.**—The laryngospasm begins suddenly in a child otherwise apparently healthy, who then grasps for breath and becomes rigid, throwing the head far back. The face becomes cyanotic, pale, or dusky; a cold sweat breaks out, and, after a brief interval, a few whistling or crowing inspirations are heard. The breathing again becomes “locked,” and presently the child, straining to get its breath, reproduces the whistling sounds. This, repeated two or three times, is followed by expiration, a vigorous cry, and the breathing again becomes established. The glottis may be completely closed, and the muscles of the thorax and diaphragm tense. If the closure be incomplete, attempts to in-breathe are laborious and noisy, as described. The effect of the strain is shown upon the heart’s action; if severe, consciousness may be lost, and the urine and feces voided involuntarily. The seizures vary in severity and in number; the milder ones may pass without much distress. The number of attacks varies from a few to thirty or forty in the twenty-four hours. If the spasm continues longer

than a minute or two, death is likely to result. Fatal terminations are unusual, however. The paroxysms may occur as readily in the daytime as in the night. There is no accompanying fever unless some other disease supervenes. Dyspeptic symptoms are nearly always present. As the disorder progresses spasmodic phenomena occur, very often in other parts of the body, the most frequent of which is the indrawing of the thumbs and the upward turning of the great toe—"carpopedal spasm." The foot is sometimes drawn up against the shin, and the hands are sometimes bent upon the forearm. The course of laryngismus stridulus is irregular, the attacks occurring at intervals of varying length and severity; the course usually, however, "runs a circuit of aggravation, climax, and diminution." The duration is uncertain, and the first attack may prove fatal in a few hours, or it may last or recur for months. The complications of laryngospasm are very numerous, involving sometimes the membranes of the brain. Oftentimes, also, there arise catarrhs of the lungs, bronchial tubes, larynx, and intestines.

**Diagnosis.**—Laryngospasm is rare in America, and so sudden and brief is the seizure and so free from disturbance the intervals that there should be little difficulty in recognizing the disorder. It is accompanied by no fever, change of voice, catarrhal symptoms, or cough; therefore there is no occasion to mistake it for croup or other organic disease of the larynx. It somewhat resembles false croup, but the clinical history is quite different.

**Prognosis.**—The prognosis varies, but is never good, a large proportion of cases ending fatally not often from the condition itself; but because of underlying and accompanying states. This is not strange when one reflects upon the enfeebled conditions of those in whom it occurs.

**Treatment.**—The important element of treatment is constitutional repair, and this is treated of at length under rickets and developmental methods. The digestion in most sufferers is conspicuously bad, and needs careful regulation. For the relief of the spasm a host of remedies are advised, the best of which are local or general counter-irritants in the shape of cold or heat, baths, and volatile substances applied to the nose. In extreme apnea relief is afforded by placing the child in a hot bath and dashing cold water on its face and chest, or a hot mustard foot-bath with ice compresses to the head and neck. Strong currents of electricity, especially faradism, to the chest and larynx may stimulate breathing. If death be imminent, blowing into the lungs is sometimes effective, or intubation may be demanded.

Tracheotomy is to be avoided. Artificial respiration is of use. The bowels should be promptly unloaded by an enema of water or glycerin; if time allows, a dose of castor oil or calomel should be given. Of depressomotor remedies, musk is said to be best; next come the bromids, valerian, and chloral hydrate, but opium should be used with great care. The dose of musk is from a grain to two grains in the syrup of lactucarium, or it may be given in the form of the tincture of musk—from five to twenty drops. During the interval tonics should be used, and it is well to bear in mind, as Jacobi points out, that in rachitis, while the heart may be of average size, the arteries are abnormally large, thus lowering blood pressure, and the circulation in the respiratory organ is slow and sluggish, tending to produce congestion and catarrh; therefore it is advisable to add cardiac tonics, as *strophanthus*, *digitalis*, or *spartein sulphate*. Many cases bear well  $\frac{1}{6}$  of a grain of codein in a day. General convulsions are liable to follow the attack, and for this the cautious inhalation of chloroform or a rectal injection of from four to eight grains of chloral hydrate may afford relief.

### COUGH

The causes of cough are those conditions capable of producing pharyngeal, laryngeal, bronchial, otitic, pleural, or pulmonic irritation, and the act of coughing usually begins in the respiratory mucous membranes. The utility of coughing is to expectorate the material which offends this surface, such as foreign bodies, mucus, or pus. Cough accompanies all forms of bronchitis, and such affections of the lungs as have a bronchitis associated with them. The cough of phthisis is due to a localized bronchial catarrh or to a laryngeal irritation. This irritation may arise in any portion of the respiratory tract. Cough is especially prominent in diseases of the pharynx and larynx. The cough of laryngeal irritation is recognizable and characteristic. The presence of a foreign body in or about the respiratory passages does not necessarily or always produce a cough, as when the sensitiveness of the parts or the general blunting of the sensibilities reduces the physiologic irritability. This occurs in certain diseases of the brain and in the stupor induced by high temperatures, especially in the infectious diseases. When in a grave pneumonia or the late stages of tuberculosis of the lungs the cough subsides, it is ground for much anxiety. Cough may also be of centric origin. Irritation of the floor of the fourth ventricle above the center of respiration excites a cough. For-

eign bodies in the meatus of the ear often excite a cough; so does disease of the ear. Infants during the first dentition are sometimes said to cough as the teeth erupt; but it is best to suspect the diagnosis of dentition cough. We do not accept that diagnosis. The cough which accompanies gastro-intestinal disturbances and which often is known to leave promptly when the condition is relieved is probably due to the secondary pharyngitis induced.

Cough may be dry or moist; constant or paroxysmal. The cough is dry when the cause of the irritation cannot be readily or promptly removed.

A dry cough is heard in the first stages of bronchitis and in phthisis. Pleurisy at first induces a short, hacking, suppressed cough; later it is superficial and is always characteristic. The cough of ear trouble or dental irritation is not unlike it. The cough of emphysema is often dry for a period, and later only ceases after the dislodgement of a small mass of mucus. The nervous cough is also dry in character. Moist cough is accompanied by the output of mucous, mucopurulent, or bloody sputum.

A constant cough implies persistence of the cause. A paroxysmal cough is one in which the irritation recurs, or the resistance is limited and may be of reflex or central causation.

If accompanied by vomiting or retching, it is a ground for suspicion of pertussis or phthisis. Pertussis is evidenced by paroxysms of coughing which are often followed by vomiting or retching, and the sufferers also exhibit a congested and anxious appearance of the face, with staring eyes, clutching at the side, etc., and followed by a long-drawn forceful inspiration or "whoop."

### ACUTE BRONCHITIS

**Definition.**—Bronchitis is an inflammation of the bronchial mucous membrane of the large and small tubes, due to many causes, and is rather symptomatic of many conditions than a disease *per se*. Capillary bronchitis, or a catarrh of the smallest tubes, is no longer used as a division of this disorder, but truly represents a type of bronchopneumonia. These final ramifications can scarcely be affected without also involving the alveoli. The only practical division is into mild and severe cases of bronchitis.

**Etiology.**—Acute bronchitis is common in children as the result of cold and exposure. It occurs most frequently late in the autumn and early in the spring, when changeable weather is prevalent. Bron-

chitis also accompanies a large number of infectious diseases, especially in children, in whom the bronchial mucous membrane is peculiarly susceptible to congestion, as in typhoid fever, measles, whooping-cough, influenza, diphtheria, and scarlatina. In infants it is also a common accompaniment of dentition, where there is a lowered condition of nutrition present, as in rachitis. Here it is more likely to be the feebleness of the individuals which renders them extremely susceptible. Irritating gases or substances introduced into the bronchi mechanically induce a bronchial catarrh; also any form of lung degeneration, notably tuberculosis. Bronchitis is also a common symptom of septicemia, and a special variety has been pointed out coexisting with putrid diarrhea. It is seen in diseases of the kidneys. Frequently recurring bronchitis is very likely to be associated with enlarged bronchial glands. No one organism can be claimed as the specific cause of this disorder. Most commonly, however, in the late autumn, the early spring or the winter months, the influenza bacillus plays the causative rôle. It may be found in pure culture, or associated with other organisms.

**Pathology.**—The anatomic changes in acute bronchitis are practically the same, from whatever cause. Owing to the swelling of the mucous membrane, the lumina of the trachea and bronchi become smaller. There is temporary functional arrest of the action of the mucous glands, with subsequent increase in their activity.

As the congestion diminishes desquamation of the ciliated epithelium in the mucosa and swelling of the submucosa, with infiltration of the leukocytes, take place. The pathologic changes, as a rule, are confined to the mucous membrane, producing no change beneath it, unless the case is protracted, when there may be slight thickening of the walls. Should the smaller bronchi be affected, occlusion may result, with collapse of the alveoli supplied by them; hence supervenes a general collapse, more particularly in infants, where the catarrhal products there heaped together produce unusual troubles—emphysema or alveolar catarrh. During the earlier stages, when the cough is spoken of as “tight,” the epithelium after the primary congestion accumulates along with little moisture, and as these cells are not then reproduced, they do not readily come away in the sputum.

The sputum consists of cellular débris, of mucous plugs secreted from the glands in the bronchial walls, and of mucus which has been formed in the epithelial cells themselves, together with pus cocci.

Should there be intense congestion, the mucus may also become blood-streaked, and on examination of the submucosa ecchymotic

spots will be found. The changes which sometimes take place in the character of the secretion have led to the subclassification into bronchorrhœa serosa, bronchitis fœtida, etc.

The lymphatic glands at the root of the lung become enlarged, particularly in infants and young children—a point of great importance. These are liable to remain more or less engorged and to become the starting-point of subsequent attacks, and while not originally tuberculous, may readily become so. This is a large factor in the production of anemia and in delaying convalescence.

In chronic bronchitis the acute form of the disease fails to undergo resolution; the cellular infiltration of the fibrous coat continues, producing a thickening of the whole bronchus with diminution of its caliber, leading first to hypertrophy and then to atrophy and impaired elasticity and favoring the formation of dilatations. If this process continues, the infiltration leads to the formation of fibrous tissue, resulting in interstitial pneumonia, and the contraction of this produces (especially when formed in the interlobular septa) a dilatation of the tubes, known as bronchiectasis. Congenital bronchiectasis may also be observed. Chronic bronchitis is always accompanied by more or less atelectasis and emphysema.

**Symptoms.**—The first symptomatology of mild bronchitis in very young children is usually that of coryza, slight catarrh of the upper respiratory passages, some elevation of temperature— $100^{\circ}$  to  $101^{\circ}$  F.—and a hard, dry cough of more or less severity, lessened appetite, some evidence of discomfort, referred to the chest or stomach, slightly hurried respiration, and quickened pulse. The pulmonary resonance is normal; a few sibilant or sonorous rales over the region of the larger bronchi (the bifurcation), posteriorly. The stage lasts from two to three days. In general, the sonorous rales are produced in the larger tubes, the sibilant ones in the smaller. When the mucus becomes more moist, and as recovery advances, there is a looser and less constant cough, along with increased expectoration and greater liveliness.

Expectoration is not possible in infants, and is only an acquired capacity of later years—at seven or eight. The mucus is therefore swallowed, producing more or less gastro-intestinal disturbance.

If the temperature should run above  $101^{\circ}$  F., and especially if it remains high beyond the third or fourth day, a critical search should be made for evidences of bronchopneumonia. Before this can be determined we may have a group of symptoms which are common to severer bronchitis and to the pneumonic extension—a dilatation of the

alæ nasi on inspiration, or other evidences of dyspnea, with a sinking-in of the tissues above the sternum or of the soft parts along the insertion of the diaphragm, and accompanied by increased rapidity of respiration and pulse. The temperature may become high or remain low. Temperature change in bronchitis is to be attributed to the cause rather than to the disease; there is no typical curve. If all this again mitigates in a day or two, apprehension lessens as to a deeper extension of the catarrh, even though there be pronounced nervous phenomena with grunting expiration and evidence of much pain on coughing. Anxiety is caused by a protraction of these symptoms; particularly if there be more or less temperature elevation, with an increased proportion of respiration over pulse-rate, a tendency for the infant or child to become apathetic, along with a cool, moist skin, we may anticipate finding evidences of bronchopneumonia upon exploration of the chest.

**Diagnosis.**—The diagnosis of bronchitis is obvious enough; a cough with bronchial secretion is not to be mistaken, but, nevertheless, the condition should be carefully differentiated from pharyngitis and bronchopneumonia. One should also strive to make an etiologic diagnosis, for it is certainly most helpful from the standpoints of prognosis and treatment to be able to say: "This is a bronchitis in epidemic influenza;" "this is simply symptomatic of measles;" or "this is bronchitis in a rachitic subject." Rales may or may not be heard at first and may be few or many on one or both sides of the chest. Later, when these become moist and more numerous, they are more readily distinguished. In a simple bronchitis, of howsoever great severity, there is no modification of the percussion-note, and in cases of circumscribed pneumonic consolidation there may be very little. The presence of bronchophony and bronchial breathing will aid us here. In bronchitis, as in bronchial asthma, we have noted frequently that the signs are at one time more marked over one lung, and at another period over the other.

**Prognosis.**—The prognosis depends upon the cause which has produced the disease, and must be estimated in the light of the previous malady. The duration of a mild bronchitis is usually three or four days, and even when quite severe, rarely lasts more than a week or a week and a half. That which follows pertussis may last indefinitely; so in measles. The situation of the affection in the smaller or larger tubes is not of uniform significance in estimating duration. If the bronchial glands are enlarged, we may fear considerable protraction and ready recurrence of bronchitis, and a danger of tubercular infection. If the child is vigorous and appears well, the catarrh is easily thrown off and

the disease ends promptly. If of lowered vitality, the disease lingers, from whatsoever antecedent cause. Constitutional defects, as rickets, are most unfavorable; so, indeed, is organic damage, such as cardio-valvular disease.

Bronchiectasis, or acute dilatation of the bronchi, may occur, due to inflammatory softening of the walls of the lesser tubes. Aëro-emphysema is commonly associated with these dilated tubules. Hence will be found hyperresonant areas, with prolonged expiratory murmur.

Collapse of the lung may take place occasionally during the course of a bronchial catarrh. The symptoms of collapse are not always definite, but are usually a distressful cyanosis, excited struggles for breath, and possibly convulsions and marked asphyxia.

The physical signs are not so clear. Percussion sounds are rendered obscure by closely associated areas of emphysema (dilatations), with collapsed bronchi and pneumonic patches. The respiratory murmur over the collapsed area is weak.

**Treatment.**—This subject should be viewed carefully from several angles. It should consider the type of infecting organism, or the nature of the underlying disease. Again, the constitution of the patient should influence our treatment. Is he strong or weak? Is he the subject of some constitutional disease (diathesis), or of some cardiac or renal affection. Once more, it should be recognized that most cases of bronchitis are of infectious nature. Therefore, it becomes necessary to limit the danger of infection to others and to himself. This involves the principles of asepsis and antisepsis. Bronchitis, like bronchopneumonia, is "notorious for the bad company that it keeps." Another indication, then, is to prevent complications and sequelæ. To all thoughtful students, we commend the careful chapter on this subject by the late John H. Musser. (Practical Treatment.—Musser and Kelly.)

No matter what views we may entertain concerning the ambulant treatment of cases of mild bronchitis in adults, every infant or child suffering with bronchitis should be put to bed. The room should be cool and well ventilated, and sunlight should be admitted freely. The principles of asepsis suggest such treatment of his nares as we have mentioned in our chapter on coryza. Protection of the ears, or the introduction into the auditory canals of cotton moistened with a 2 per cent. solution of carbolic acid in glycerine, are prophylactic measures that appeal to us. The eyes should be cleansed with boric acid solution at three-hour intervals. Nasal secretions and sputum, if the child is old enough to expectorate, should be caught in gauze and promptly destroyed. Castor oil is the best laxative at this stage,

We firmly believe that the universal employment of such treatment would greatly lessen the dangers to the sick individual, and the menace to the community in which he dwells.

Is it possible to abort the attack? Sometimes, and in most instances, the period of illness may be shortened appreciably. The old time hot mustard foot bath initiates a good beginning of abortive treatment. Hot drinks may aid, and in all cases fluids should be given freely throughout the disease. Diaphoretics may prove useful—particularly the spirits of nitrous ether and Dover's powder. If there is headache, or if epidemic influenza be suspected, the coal-tar products possess an almost specific action. On the other hand, if there is much coryza, belladonna may prove a valuable drug. We have expressed ourselves concerning quinine, and are glad to find that Musser viewed it as a valuable drug in the early stages of bronchitis. Alkalies in large doses are sometimes used in this stage. Sodium bicarbonate, potassium citrate, and ammonium iodide are good examples of this class of drugs.

Local applications to the chest are of signal service. The ones that we employ most often are iodine (5 per cent.) in oily solution; guaiacol (5 to 10 per cent.) in lanoline; and mustard. We prefer to refrain from the use of mustard unless there is pronounced soreness in the region of the trachea and sternum. Inhalations of medicated steam may be of much service. Early we use the tincture of benzoin; and later, as a more stimulating inhalant, creosote with oil of eucalyptus and turpentine. The steam may be generated in a croup kettle or in a steam inhaler. In a severe case, the patient may be placed in a croup tent, and the steam made to pass under it.

Secretion having been established, stimulating expectorants are in order. Chief among these are the ammonium salts. Ammonium chloride is of benefit not only as an expectorant, but also as a stomachic. The carbonate of ammonium is seldom needed; but it does not tend to disturb the stomach of the child as it often does that of the adult. One may adopt Osler's suggestion and use aromatic spirits of ammonia. The ammonium salts should be used in sufficient dosage: at least 2 to 5 grains to the dose. Ammonium iodide is another valuable drug, though it should be administered in smaller doses than the chloride.

Other valuable expectorants are terpene hydrate (gr. i-ii); terebene; oil of tar (2 to 5 drops on a lump of sugar); creosote, and creosote carbonate (creosotal). If the cough shows a tendency to persist, creosote or creosotal and cod-liver oil are the most useful drugs. Convalescence demands tonic treatment.

Vaccines may prove of service in some cases, and will probably

prove of more service in the near future. Holt records a remarkable result in chronic influenzal bronchitis from the employment of an autogenous vaccine.

Let us epitomize a rational treatment:

a. The patient is put to bed as described, after he has had a mustard foot bath, and his chest has been rubbed with iodine and vasogen (5 per cent.) or guiacol and lanoline (5 per cent.). His nose is sprayed and his ears protected.

b. He is then given a dose of castor oil.

c. Favorite prescriptions are then:

R. Spt. æther. nitros. . . . . fl. ʒij  
Liquor potass. citrat. . . . . fl. ʒj  
Glycerine . . . . . fl. ʒij  
Aq. cinnamom . . . . . q.s. ad. ft. fl. ʒiij  
M.

SIG.—fl. ʒi every three hours.

or,

R. Pulv. opii et ipecac. . . . . gr. ij-iiij  
Acetphenetidin. . . . . gr. xij  
Phenol. salicylat. (or hexamethylenamin). . . . . gr. xij  
Sacchari. alb. . . . . gr. xij

Div. in chrt. xij.

or, to older children:

R. Camphor. monobrom. . . . . gr. xij  
Acetphenetidin . . . . . gr. xvij-xxiv  
Cinchonid. salicylat. . . . . gr. xij  
Acetyl salicylic acid . . . . . gr. xxxvi  
Pulv. glycyrrh . . . . . q.s.

Div. in capsul. xij.

SIG.—One capsule every three hours.

d. Medicated steam is produced in the apartment for one hour at a time, and three or four times a day.

e. Useful expectorants are:

R. Codein. sulphat. . . . . gr. ʒ  
Ammon. chlorid . . . . . gr. xlviij  
Syrup. Pruni. Virgin. . . . . fl. ʒij  
Syrupi simplic . . . . . q.s. ad. ft. fl. ʒiij

M. et solv.

SIG.—fl. ʒi every three hours.

or,

R. Ammon. iodid. . . . . gr. xxiv-ʒj  
Ammon. chlorid. . . . . gr. xlviij-ʒj  
Syrup. toltian . . . . . fl. ʒj  
Syrup. simplic . . . . . q.s. ad. ft. fl. ʒiij

M. et solve.

SIG.—fl. ʒi every three hours.

or, to older children:

R. Heroin. . . . . gr. ʒ  
Creosotal . . . . . mxl-fl. ʒj  
Terpin. hydrat. . . . . gr. xl  
Pulv. glycyrrh . . . . . q.s.

Div. in capsul. xx.

SIG.—One capsule every three or four hours.

*f.* Tonics should be given during convalescence. Combinations of strychnine, quinine and iron (as in the well-known elixir); nuxvomica before meals, and Fowler's solution after meals; the syrup of the iodide of iron, and cod-liver oil, are tonics of recognized value. A short residence in some pine-forest district, or at the sea-shore, will often hasten a return of health. Children of tuberculous parentage, or with latent tuberculosis, should receive the proper climatic treatment.

### CHRONIC BRONCHITIS

Chronic bronchitis or subacute bronchial catarrh is occasionally encountered when the child has either had repeated exposure and recurrent attacks of bronchitis or is so weakened in health as to be unable to acquire full restoration after a fresh attack. Sometimes it is not possible to account satisfactorily for the condition, though there will usually be a history of many rapidly following attacks of acute bronchitis or an unusual susceptibility. This last may be inherent or the result of specific disease, as pertussis, measles, or diphtheria. The rachitic child is peculiarly susceptible to bronchitis and irritative cough; also members of tubercular families or the so-called scrofulous children. Adenoids are very often present in these subjects and unquestionably furnish a tissue *minores resistentiæ*.

Season often has a marked influence. The children may remain perfectly well during the summer season only to contract a cold in the autumn, and then one after another until the so-called "winter cough" is firmly established.

The **symptoms** are troublesome cough, expectoration, moist or dry rales, generally in both lungs (if fine rales, they are in the bases usually), absence of fever, and unimpaired resonance and, usually, emphysema.

**Complications of Bronchitis.**—*Pharyngeal catarrh* is a frequent cause of aggravated cough and demands local treatment, such as frequent washings with nitrate of silver (weak solution), alkaline antiseptic spray, extract of hamamelis, or applications of iodine, potassium iodid, and glycerin solutions, tannic acid in glycerin, or menthol camphorate in liquid petrolatum.

*Fibrinous Bronchitis.*—A false membrane may be formed, resembling diphtheria. Use inhalations of steam, turpentine, benzoin, lime-water, chlorid of ammonia, fumigations of calomel, ten to fifteen grains every few hours under a tent. Internally give potassium iodid, large doses.

*Asthma* occurs along with or following bronchitis, and is frequently

due to peribronchitis and emphysema; hence potassium iodid is indicated, with an opiate at night. One should always eliminate the possibility of thymic asthma. Cleansing the nares is useful, and it is well always to remove all obstruction or hypertrophies. For the asthmatic attacks: tincture of lobelia, two or three drops; fluid extract quebracho or grindelia,  $\frac{1}{2}$  to one dram of each, will often relieve dyspnea (Jacobi). Fluid extract euphorbia pilulifera, increasing doses, is a very useful antispasmodic. Night cough and night irritation are relieved by bedtime doses of bicarbonate of soda or carbonate of magnesia and codein and hyoscyamus or laxatives of castor oil or cascara.

*Emphysema* in children presents an easier diagnosis than in adults: the lung tissues are more elastic; cellular activity of the alveoli is more active and more easily influenced by nutrition and by remedies, respiratory exercises, and blood repair. To induce sneezing is of value. Nebulized expectorants and resinous preparations are useful.

**Diagnosis.**—From fibroid phthisis the distinguishing points are absence of dullness and of such rales as point to dilatation of bronchi or consolidation, normal vocal fremitus and resonance, and one or more of the occasional outpourings of offensive smelling mucopus which accompany fibrophthisis.

*Additional Suggestions as to Treatment of Bronchitis and its Complications.*—Certain authorities recommend bicarbonate of soda, ten to fifty grains a day, with one grain of ipecac; ten to fifteen grains of ammonium chlorid a day, or two to five grains every three hours; apomorphin, in older children,  $\frac{1}{150}$  of a grain every two or three hours. or potassium iodid to loosen a "tight cough"; terpin hydrate, in doses of  $\frac{1}{4}$  to  $\frac{1}{2}$  grain, frequently repeated, especially in the chronic form; also terebene, ten- to twenty-drop doses. The complication of asthma is benefited by iodid of potassium, increasing doses to toxic effects, then slowly decrease and stop. Accumulated mucus may demand an emetic; if danger of suffocation or great effusion, frequent changes of position. If cyanosis arises and the voice is not heard, it is imperative to make the child cry—slapping with a wet cloth, swinging about, faradic current, etc. If cough is irritating, small doses of opium, codein, or extract hyoscyamus, the opiate especially, at night. Chlorid of ammonia evaporated on a hot stove, turpentine atomized, and compound syrup of white pine are useful expectorants; compressed air and oxygen are of great help in dyspnea and during tardy convalescence. In a case of chronic influenzal bronchitis under Dr. Holt's care, an autogenous vaccine yielded very phenomenal curative results.

## BRONCHIAL ASTHMA

Bronchial or spasmodic asthma is a form of paroxysmal dyspnea along with wheezing respiration occurring in sharp attacks; but perfect health is enjoyed between the intervals. It occurs quite frequently in childhood, and rather oftener in the upper than in the lower walks of life. Most writers agree that it is a neurosis resulting from disturbed innervation of the pneumogastric or its ramifications or the vasomotor nerves, and causing a spasm of the muscles of the air-passages. Hay-fever is a form of reflex asthma which accompanies marked coryza. The affection seems to run in certain families, especially those of irritable and unstable nervous equilibrium, and exciting causes are only operative when there is a predisposition to the disease. Rag-weed appears to play an important exciting rôle in the production of hay-fever. Oftentimes no definite exciting cause can be demonstrated in cases of asthma. The respiratory center in the medulla is thought by some to be irritated by vitiated blood of a wide variety of sepsis. Enlarged bronchial glands pressing upon the larger bronchi (they are really mediastinal glands) seem responsible for a few cases. Bronchitis alone or in an emphysematous lung is frequently recognized as an exciting cause. Various mechanical irritants to the upper respiratory mucous membrane, as the air of certain localities, dust of various sorts, palpable or impalpable powders, odors, as of flowers or of hay, the effluvia of animals or decomposing substances, and an endless variety of causes of most differing kinds are competent to excite an attack. The importance of adenoid vegetations has been dwelt upon. Changes in the barometric pressure and disturbances of the stomach are the most efficient instrumental causes. Asthma and eczema sometimes coexist or alternate with each other. The question of thymic asthma will be given due consideration in the chapter dealing with the status lymphaticus.

The **pathology** of bronchial asthma is not known; there are many theories, such as that it may be due to spasm of the bronchial muscles; also that the attack is due to the swelling of the bronchial mucous membrane, to a catarrh of the bronchioles, etc. Talma says that the phenomenon is a spasm of the larynx and aditus, rarely a spasm of the constrictors of the glottis, and that it is partly under voluntary control, which should be cultivated as much as possible.

**Symptoms.**—Asthmatic attacks usually occur without warning. In some there are slight premonitions, rather vague; in others there is itching of the skin, copious urination, or a slight nasal catarrh. The paroxysm generally comes on at night, the patient being awakened by

distressing dyspnea, steadily increasing, with characteristic wheezing and oppression or pain of the chest. He must sit up, and is more comfortable holding on to objects, with shoulders elevated and head thrown back, to give the muscles of respiration and their allies the utmost play. The face becomes anxious, pallid, and, later, cyanotic; the skin is moist, and respiration is loud and wheezing. The respirations are rarely much increased in number; speech is difficult; inspiration is jerky and expiration prolonged and laborious, and there is little or no thoracic expansion. The pulse is rapid and thready, there is no elevation of temperature, but in a prolonged attack it becomes subnormal, the extremities growing cold and clammy, the face livid. After the paroxysm there is usually much exhaustion, and the patient sleeps. On awakening there is little left except muscular soreness, and the return to usual health is prompt. Percussion during the paroxysm shows hyperresonance, and there is heard some diminution or prolongation of the vesicular murmur. Toward the close of the attack moist rales are distinguished, and piping, wheezing, and cooing sounds.

The **diagnosis** should not be difficult, but bronchial asthma may be confused with obstruction of the upper air-passages, foreign bodies, croup, edema of the glottis, suffocative urticaria, new growths in the larynx, tracheal stenosis, pulmonary edema, pleuritic effusion, uremia, and cardiac disease. Bearing in mind the absence of elevation of temperature will enable one to distinguish asthma from the inflammatory affections. Emphysema and asthma frequently coexist, and the one may superinduce the other. Cardiac asthma is rare in children.

The **prognosis** in young subjects is good; especially is improvement marked in some cases about the time of puberty, and if there be no serious complication or grave underlying disorder.

**Treatment.**—Most sufferers from bronchial asthma are below par in health in some direction, and these factors need special reparative treatment as well as specific medication. Some sufferers from asthma, however, are of magnificent physique, as evidenced by two personal friends of the authors, who have made themselves famous on two continents as monumental specimens of vigor. The treatment divides itself into the special medication directed to the prevention of the paroxysm and the use of remedies to relieve the suffocative attacks. It is quite impossible to enumerate here the wearisome array of remedies and measures offered for the relief of asthma, especially if we include, also, disturbances from that disease, known as hay-fever, rose cold, and the like. Wherever there can be discovered exciting causes,

the most obvious of which are nasopharyngeal obstructions, these must be removed, and thoroughly. Hypertrophied turbinated bodies, or lingual or pharyngeal tonsils, adenoid growths, nasal polypi, and catarrh of any part of the respiratory tract demand attention. Climatic and domestic conditions must be revised; diet should be modified and, as a rule, lessened. Hygiene; out-of-door life; cold sponging, always followed by a dry rub; change of climate here and there, in the choice of which there is almost no special rule; the use of certain tonics (cod-liver oil and iron are lauded) and of various alteratives; long courses of arsenic and of various sorts of alkalis—produce a fair proportion of cures. The best single remedy is potassium or sodium iodid, and perhaps the most satisfactory plan of treatment, certainly in our experience, is the following: Put the sufferer to bed, or limit his activities as much as possible; feed upon an absolute diet of skimmed milk, with peptonizing and antifermentative measures if necessary; keep the bowels open, preferably with salines (mixed ones are better than single, like Apenta water), and administer potassium iodid in rising doses, one drop of a saturated solution extra each day, until full toxic symptoms are produced; then slowly reduce in the same order to the point of tolerance, and keep at that for about a month; then slowly lessen until none is taken. In so complicated a malady as bronchial asthma, with its peculiar features, the vigorous health so often seen, which is suddenly and overwhelmingly paralyzed by intensity of distress, argues either for a profound neurosis or for diathetic conditions as yet not understood, but for which it is reasonable to hope and expect to discover a controlling remedy sooner or later. The relief of the paroxysm is also not to be predicted; some simple thing may accomplish this perfectly or powerful medicines may be demanded. Morphin, administered under the skin, especially along with a little atropin, promptly relieves most adult cases, and may be given with the utmost caution, to children; but its frequent use is, of course, not to be encouraged. Next to morphin, epinephrine represents the best remedy. Epinephrine (say  $\frac{1}{100}$  v of adrenalin chloride solution) must be used hypodermatically to get the best result. Indeed it is useless when given by the mouth. The inhalation of chloroform offers prompt and temporary relief. Chloral hydrate, given by the mouth or rectum, is better and more lasting. Nitrite of amyl or iodid of ethyl inhaled, or nitroglycerin given by the mouth, pilocarpin under the skin, the use of many pungent smokes, as of stramonium, relieve some cases. Perhaps the best-known remedy is the fumes of niter paper; and safe enough it is, especially if administered under a tent. There are various combinations put up

in the form of cigarettes, the materials of which are modestly concealed; or powdery substances to be burned, sold in the shops as infallible remedies, are quite useful. Tobacco is advocated, but is depressing to children. The nitrites are praised, but are disappointing; so are *grindelia* and *quebracho*. The best, perhaps, is the fluid extract of *euphorbia pillulifera* in increasing doses. Talma claims that patients should be systematically taught respiratory gymnastics, learning to breathe slowly and deeply. We can also assert that this treatment has done much for those under our control. Dunbar's serum has effected some wonderful cures of sufferers from hay-fever.

### PULMONARY EMPHYSEMA

Pulmonary emphysema is a condition of the lung in which air to an abnormal extent accumulates within it. The interstitial form, known also as the extravascular, gives rise to no untoward symptoms, as a rule. The vesicular variety, also known as alveolar emphysema, is of much more importance if the process be extensive, but is extremely rare in children under ten years of age. This is again divided into two varieties: (1) Compensatory emphysema, wherein the vesicles of one portion of the lung are abnormally distended in consequence of the disablement or insufficient expansion of some other part of that organ. This form of emphysema is occasionally seen in young children, the causes being predisposition, enfeeblement, lowered nutrition, and locally mechanical obstruction, which produces increased vesicular pressure. Excessive coughing in pertussis produces violent respiratory efforts, inducing air-bubbles to escape into the interlobular tissue, as well as hyperdistention of the alveoli. In most cases, however, complicating acute bronchitis and pertussis the emphysema is but temporary, and small structural change is left. (2) Substantive emphysema is a chronic and scarcely curable malady, characterized by abnormal distention of the air vesicles along with structural changes in their walls. It is almost never seen in children under ten years of age, but is more frequently encountered during adolescence. It would seem to be due in part to hereditary tendency. Bronchial catarrh inducing swollen mucous membranes, and much pressure in the bronchioles due to sticky mucus, induce typical collapse, and the neighboring lobules endeavor to do double work and become hyperdistended.

Again, during enforced respiratory efforts with a closed glottis, as in violent paroxysmal coughing, the air is driven back and becomes lodged in the less resisting areas, as the apices and anterior borders of the lungs. In any event there needs to be some inherent feebleness or

degenerative change to account for the anatomic alteration. This may be a chronic inflammation, a pneumonitis attended with the production of fibrinous tissue, along with an atrophy of the normal parenchyma. The postmortem changes are much the same as in the emphysema of adults. There is usually some hypertrophy of the right ventricle of the heart and perhaps secondary dilatation. The symptoms of emphysema in young children are oftentimes negative, and when present, are like, but milder than, those in adults. Dyspnea is one of the most common symptoms, at first only noticed upon exertion, and later more severe upon slight catarrhal disturbances of either the nose or the nasopharynx, the bronchi, or digestive organs. Cough is readily excited, especially in winter, and asthma often arises. Children rarely exhibit the barrel-shaped chest of emphysema, but they do sometimes show an increased depth anteroposteriorly and rigidity. Percussion gives signs of value; if very light percussion is practised, particularly in the supraclavicular fossa and over the cardiac area, but the thoracic walls of children are so elastic and the organs so small, that transmission of resonance is favored from other parts. Auscultation reveals a low-pitched respiratory murmur with prolonged expiration; the second sound of the heart may be accentuated.

**The diagnosis** is made upon the history of chronic bronchitis, or other of the causes mentioned, the symptoms and the physical signs.

**Prognosis.**—Recovery may be looked for if the disturbance has not continued too long. Emphysema itself does not imperil life except as a complicating factor in other diseases. On the whole, emphysema in young subjects offers a much better outlook than in later life.

**Treatment.**—The treatment is mainly prophylactic; exciting causes should be guarded against and receive prolonged treatment. Attention to the skin by cool bathings and thorough rubbing must be faithfully and continuously given, and woolen underwear worn. The digestive organs must be guarded from disturbance with unusual faithfulness. Many tonics are of value, of which iron has a suitable place, especially in the form of Basham's mixture. Nux vomica is of efficacy, and arsenic, usually in the form of Fowler's solution, more so. In the chronic bronchitis which so frequently coexists, iodid of potassium is of special value. Next come the resinous preparations, then terebene and guaiacol. Cod-liver oil is a reliable tonic.

#### ACUTE BRONCHOPNEUMONIA

Bronchopneumonia—also called catarrhal pneumonia, lobular pneumonia, capillary bronchitis, etc.—is an inflammatory disease of the

terminal bronchioles and pulmonary air vesicles, affecting the lobules in scattered areas. The disorder is in the main a catarrhal inflammation of the bronchioles and air-sacs, although the peribronchial and inter-ventricular tissues are also involved. The disease often proves fatal in

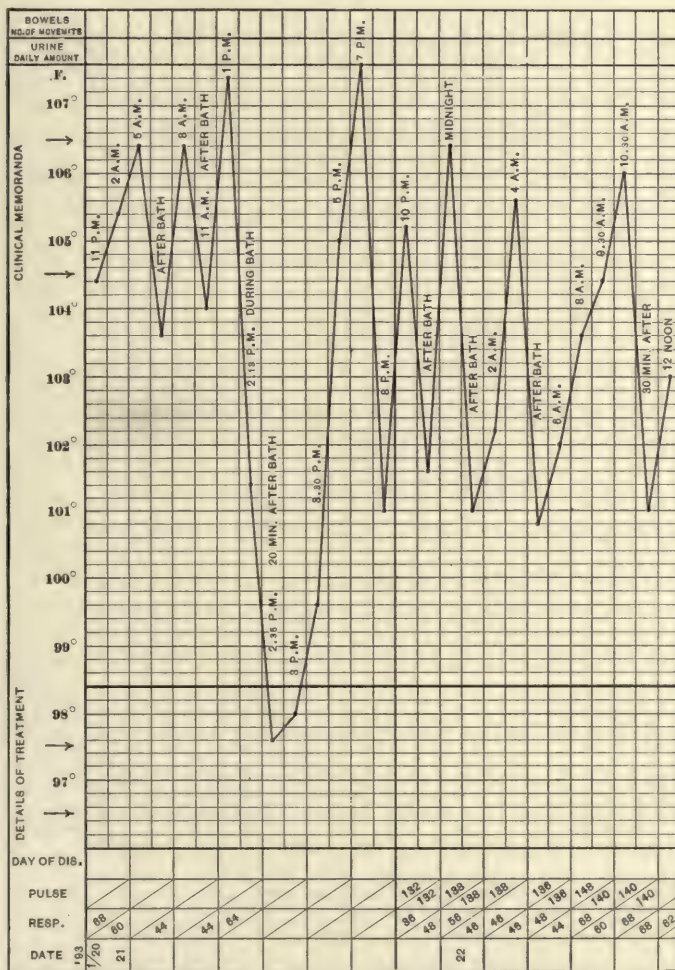


FIG. 54.—FATAL CASE OF BRONCHO-PNEUMONIA WITH HYPERPYREXIA. (MAXIMUM TEMPERATURE=108.2°). THE PATIENT WAS A RACHITIC NEGRO INFANT, TEN MONTHS OLD.—(Philadelphia General Hospital.)

a few days, or it may linger in a chronic form, leading to secondary changes or inducing a tendency to tuberculous infection. We think it a mistake to view bronchopneumonia as a single disease entity. Clinically as well as etiologically, one views rather a group of diseases. We agree with Holt that one may recognize: 1. The acute congestive

type of early infancy; 2. the capillary bronchitis type; 3. the ordinary type, usually affecting both lungs, and most often the bases; 4. the mild type; 5. the massive type, which may become lobar; 6. the abortive type; 7. the caseous or tuberculous type; 8. the white pneumonia of the new-born; 9. hypostatic pneumonia; 10. aspiration (or inspiration) pneumonia.

**Etiology.**—The primary cause is usually bronchitis, either the simple catarrhal form or that which accompanies or follows infectious processes, especially measles, whooping cough, diphtheria, or tubercu-

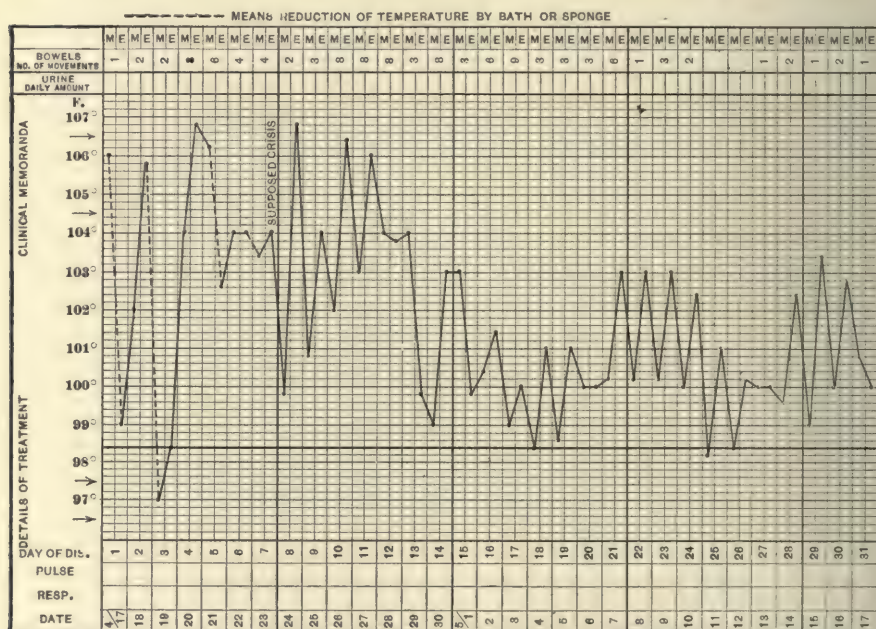


FIG. 55.—BRONCHO-PNEUMONIA OF SIX WEEKS DURATION IN A SEVENTEEN MONTH'S OLD BABY. MARKED HYPERPYREXIA.—(*Philadelphia General Hospital.*)

losis. Bronchopneumonia is in nearly all cases a secondary disease; it may arise as a primary disease, however, or from an untraceable cause, or as the result of irritants, mechanical or gaseous, entering through the mouth, nose, or respiratory passages. In the bronchopneumonia of diphtheria the streptococcus is the usual cause. In the new-born it may result from inhalations of the maternal secretions during birth. In many cases the specific cause is the pneumococcus, the tubercle bacillus, or in certain instances the staphylococcus aureus and albus or streptococcus pyogenes, or the influenza bacillus. Secondary cases are usually due to a mixed infection. Most fatal cases occur under two years of age, especially during primary dentition. Broncho-

pneumonia is also a deadly complication of the infectious diarrheas of the summer season. Pneumonias stand next to diarrheas as a cause of infantile mortality, and the lobular is much more common than the lobar form. Infants show a marked tendency to catarrhal processes. Depraved vitality is a powerful predisposing cause, the result of unwholesome environment and diet and improper care. Especially is this noticeable during the prevalence of epidemics of measles, diphtheria, whooping-cough, and influenza, during convalescence from which sufficient care is too rarely exercised. Season has much to do with the prevalence of this disease, it being by far the more frequent during the winter and spring, and especially if the weather be changeable from wet to dry. Steady cold is rather favorable to immunity from catarrhal pneumonia, but during damp summers it is liable to prevail. Lastly, it should probably be said that in most cases of bronchopneumonia, hypostasis and aspiration have both played etiologic rôles. This conception is of vast value in intelligent treatment as well as in prevention.

**Pathology.**—The essential lesion in bronchopneumonia is an inflammation of the walls of the terminal bronchi, bronchioles, and the adjacent alveoli, with the rapid casting off of epithelial cells, one after another, along with a few leukocytes or red corpuscles accumulating within. As the process continues there is an increase in cellular desquamation, with outpouring of mucus, filling up the tubes and air vesicles; thus centers of consolidation are formed in different parts of the lung, at times only attacking small portions, and again invading large areas irregularly (see varieties of bronchopneumonia). Both lungs are usually attacked, sometimes so extensively as to involve a whole lobe (massive type). Bronchopneumonia develops by the regular invasion of successive portions of the lung, and resolution takes place in the same gradual manner. The mottled appearance seen on inspecting an infected lung may be due to areas of red and gray hepatization in close proximity. Absorption of the fibrinous exudate takes place more readily than of the cellular elements; and hence in bronchopneumonia, where the inflammatory products are mostly cellular, they are more slowly resolved than in lobar pneumonia, in which they are mostly fibrinous. The process begins in the terminal bronchi and extends into the bronchioles and air-sacs, either by inflammation, following the epithelial lining, or mechanically, by violent inspiratory efforts induced by coughing. Thus the smaller tubes become plugged up, producing atelectasis, even where the act of expiration forces the air out of the cell, and inspiration being a weaker

act, they fail to become or are only slowly refilled. Once the vesicles become collapsed congestion takes place, which is also partly mechanical, inflammation results, and the temperature goes up. Parts of the lungs which are not hepatized are congested and edematous, and the air-spaces in the alveoli are encroached upon by the congested blood-vessels and epithelium.

On microscopic examination the bronchioles will be found filled with an exudate containing leukocytes and epithelial cells; occasionally, also, blood-corpuscles. The alveoli in the immediate neighborhood of the affected bronchus will be more completely filled with the exudate than the outlying ones. The capillaries in the walls of the bronchi are somewhat distended, and the general appearances of an interstitial inflammation are present.

These areas of collapse are mostly symmetric, occurring in the posterior borders of both lower lobes, and sometimes in the upper ones, and may arise during the acute stage or when the pneumonic process is more definitely established. There are no clearly marked stages in bronchopneumonia as in lobar pneumonia, and all of them may appear in different areas of the same lung. Nearby these zones of congestion the bronchi frequently become dilated, owing to the weakened condition of the bronchial walls; yet this condition is liable to disappear entirely upon recovery. Emphysema, usually vesicular, frequently arises in the course of bronchopneumonia and more commonly in the upper lobes; but it may be wide-spread, and is more common after whooping-cough. There may be interstitial emphysema also, caused by the rupture of air vesicles lifting the pleura or extending between the lobules. Bronchopneumonia may persist, the proliferative cells taking part in the formation of new connective tissue, causing persistent thickening. This occurs especially after other than first attacks. The walls of the bronchi and the peribronchitic tissue are at times subject to a persistent thickening and fibrinous formation, producing a chronic bronchopneumonia. The cicatricial tissue surrounding the bronchi causes an increased dilatation in the walls already weakened, and there follows saccular as well as fusiform dilatation.

The macroscopic appearances usually show a tendency to lobular limitation, these lobules being raised somewhat from the surface and exhibiting alternations of dark-red or grayish color. Sometimes a whole lobe may be affected, and then it is very like the appearance of croupous pneumonia. Next to the consolidated lobules there is sometimes seen emphysema, the one overlying the other. On the

lung being cut, the various lesions are seen most beautifully, areas of atelectasis and of peribronchitic congestion adjoining the smaller bronchi (from some of which issues mucus or mucopus) and emphysematous patches. The air vesicles are themselves sometimes inflamed and found filled with red blood-cells. In the very acute cases extravasation of blood occurs into the alveoli just beneath the pleura. Adjoining the consolidated areas there is usually pleurisy, especially over considerable areas of consolidation, binding the lung to the chest-walls. There is also occasionally a moderate amount of exudation into the pleural cavity. Indeed, some pleuritis probably always occurs over the affected lung area.

The causes of death in pneumonia of children are (1) exhaustion, (2) acute toxemias, and (3) complications. Exhaustion is the most common and is to be met by sedulous attention to hygiene and diet. Nursing infants should be spared overofficious attention. The complications are most liable to cause death, particularly otitis and empyema.

**Symptoms.**—Bronchopneumonia, as has been said, is almost always a secondary disease, and it is liable to arise in the course of a bronchitis or of some one of the contagious diseases—measles, scarlatina, whooping-cough, influenza, and diphtheria especially. There is then to be recognized some increment of fever, an increased pulse rate, and especially a difficulty in breathing. The fever is at its height in from three to five days, and usually quite irregular in course, above  $102^{\circ}$  up to  $104^{\circ}$  or  $105^{\circ}$  F., or in rare cases even higher. The height of this becomes a fair index to the virulence of the disease. Deaths with low temperature occur only in very feeble children (adynamic pneumonia). The rise and fall on the temperature-chart is gradual, and feverishness persists for a considerable time. The respirations are usually hurried, disproportionately so. The respiration is, however, more labored than panting, a valuable sign in differentiating between bronchopneumonia and the croupous form. The “expiratory grunt” is also in evidence. The pulse becomes greatly increased (125 to 150), and the ratio between pulse and respiration is from one to two, or even less. The cough is usually short and hacking, often less distressful than in a severe bronchitis, and more consciously controlled. The cough is a valuable index of the vigor of the child and the progress of the case. When this becomes feeble, it is a sign of respiratory and other organic failure. The pain felt is less in front, as in bronchitis, and more in the side. The *alæ* of the nose are seen to dilate, especially in little children. When atelectasis takes place, the dyspnea becomes more marked and the expiration “grunting”; the skin becomes livid and dusky, and, as we

have often had occasion to remark, the characteristic symptom here is increased relaxation and leakiness of the skin. As the symptoms become severe the child remains more and more quiet, holding its head well back and supporting itself by the hands; it is a characteristic feature that the worse the malady, the more submissive is the child. Expectoration is liable to be mucopurulent, but infants and children under seven or eight years swallow and conceal this. Appetite is generally lost, but thirst is excessive. The suckling of infants is difficult and incomplete, on account of the dyspnea. Strength is rapidly lost, and somnolence is a very evil sign. Vomiting may occur at the outset, and diarrhea is a bad complication. Irregular fever with high pulse-rate indicates a protracted course, and possibly chronicity. The heart must be carefully watched; a feeble first sound is of gloomy import, though death is more likely to result from respiratory than cardiac failure. In the beginning there are the usual signs of bronchitis—first dry and later moist rales, without dullness. Contrary to popular belief, the physical signs of catarrhal pneumonia are very distinctive, provided that one has proper conceptions concerning the multiform clinical aspects of this disease (see varieties of catarrhal pneumonia) and also knows what to expect in his physical examination. To await the occurrence of well marked consolidation, before making a diagnosis, however, means that many infants die with their cases misunderstood. Again, one must be prepared for rapid changes in the physical signs and changes in their location. These things are characteristic of bronchopneumonia. We shall attempt to enumerate the most important physical signs in our own experience:

**Inspection.**—Cyanosis; play of the *alæ nasi*; marked accentuation of the nasolabial line; recession of the suprasternal notch and xiphoid in inspiration; preponderance of abdominal respiration, with a thoracic movement that is chiefly up and down. Differences in expansion of the two sides of the chest are not observed, unless the process be a massive one and confined to one lung.

**Palpation.**—As Osler has pointed out, this may be misleading; but in many cases it is very useful. Increased rhonchial fremitus may be noted, even the mother calling one's attention to the "rattle on the chest." In the acute congestive or capillary bronchitis types this may be general, or it may be more marked over the affected area. Tactile fremitus may be much accentuated during crying. Differences in expansion may be felt that have not been detected in inspection.

**Percussion.**—We quite agree with Holt upon the value of tympany as a physical sign. If we were asked: Which is the more important

physical sign, tympany or impaired resonance, we should be inclined to reply tympany. For in the acute congestive type and the capillary bronchitis type, impaired resonance does not occur; but the chest may be universally tympanitic. Again, tympany is an earlier sign than impaired resonance, often betraying, with accuracy, the portion of the lung that will be dull upon the succeeding day. And still further, tympanitic areas are nearly always discoverable in the neighborhood of dull or flat areas. (See Pathology.) Impaired resonance, dullness, or actual flatness, however, may be detected in many cases and is a sign of value. Light percussion should be used to elicit it, for in the main, we are dealing with superficial, not lobar areas of consolidation. We are not surprised that such areas of impaired resonance are usually small, that they are more commonly found near the bases, and that they rarely invade the regions of the axillary lines. These things our study of pathology would lead us to expect, and only in the massive types do we find a lobe universally dull.

**Auscultation.**—An early important sign is weak breathing—so weak at the affected base that students often think the well lung is the pneumonic one. On the following day, however, bronchovesicular breathing is likely to have succeeded the weak breathing in the same region. Only when there are large areas of consolidation, does one hear typical bronchial breathing. If the infant cries, one may often detect broncophony where the bronchovesicular breathing is most marked. A stethoscope with a small bell is needed for this study. As an early sign, the high pitched sibilant rales (consonant rales of the English) are most valuable, almost pathognomonic. They may be heard in many parts of the chest (acute congestive and capillary bronchitis types) or only over the affected region (usually the bases). In addition to these, we have the auscultatory signs of bronchitis. Later on subcrepitant and mucous rales are heard.

**Complications and Sequelæ.**—Pulmonary collapse is less a complication than a feature to be expected. Sometimes it is so extreme as to warrant the use of the old term "suffocative catarrh." Pleurisy is a frequent but only rarely a troublesome complication unless an empyema develop. The most serious sequela is tuberculosis; indeed, the process may have been tuberculous from the start. Bronchopneumonia renders a person peculiarly susceptible to this poison. Meningeal symptoms sometimes arise toward the end of the disorder, probably due to hyperemia of the meninges or to toxemia. Chronic or continued pneumonic process is a serious feature not uncommon. Cases may go on to rapid or slow resolution and yet recovery be com-

plete and thorough. A form of continued catarrh of the alveolæ is described by Douglas Powell, usually seen in the apices of the lungs. This begins as a proliferation of the alveolar epithelium. In the milder cases the inflammatory products are expectorated, leaving the alveolar walls undamaged; when the inflammatory process is greater, the proliferated epithelium and the leukocytes are sufficient to block the alveoli completely. The exudate undergoes degeneration, and may be partially absorbed and partly expectorated, but the alveolar walls have been damaged and collapse results from their agglutination. The inspissated products may remain a long time, or the fibrous stroma may become involved and a hyperplasia result, and ultimately a fibrosis takes place of the affected area. Such cases may linger long in this condition or fall into serious states, or die of exhaustion or some intercurrent malady, especially tuberculosis. Other forms of bronchopneumonia suffer relapses or recurrences and result in a chronic interstitial pneumonia.

Associated lesions of the lung are enlarged bronchial glands, more or less emphysema in almost all cases, gangrene rarely, and abscess. (Most abscesses, however, are probably interlobar empyemata). Middle ear disease is the most common complication, and should never be forgotten. Next in the order of frequency is empyema.

**Diagnosis.**—It is important to examine every case of bronchitis for the intercurrent of a possible pneumonia, especially in the infectious diseases. This may be recognized by sudden increase of fever, acceleration of pulse and respiration, and the physical signs described. Catarrhal pneumonia may exist without signs of consolidation. If the disease is first seen in a well-developed state, it may readily be confused with croupous pneumonia, but along with the history of the case, scattered areas of dullness, tympany and râles will help to distinguish it. Pleurisy often accompanies bronchopneumonia and confuses the physical signs. When gastro-intestinal disorders or nervous symptoms are marked features, the pneumonia may escape attention unless searched for. Here the changed respiration and pulse-rates should be indications.

The **prognosis** is guardedly bad, the average mortality being nearly 50 per cent. under five years of age, and less over that period. In private practice (Holt) the mortality is from 10 to 30 per cent. The cases which follow whooping-cough are most serious; also those complicated by previous marked debility. The duration is usually from two to three weeks, mild cases terminating sooner. The symptoms to be considered in making a prognosis are the height and course

of the temperature, the occurrence of nervous or toxic phenomena, the state of the digestive organs, the presence of cyanosis, and the extent and character of the diseased areas, and also whether or not the process follows or complicates an infectious disease.

**Treatment.**—The chief element of treatment is prevention, bronchopneumonia being essentially a secondary disease and controllable in most instances by proper care. Prophylactic measures of importance have been mentioned in our chapters on coryza and bronchitis. The measures used should be early applied. Any case of bronchitis may be complicated by more or less pneumonia. (For treatment see separate article on Treatment of Pneumonia.)

### CHRONIC BRONCHOPNEUMONIA

Chronic bronchopneumonia, also called fibroid phthisis, is a condition of the connective-tissue framework of the lungs following upon other diseases. The condition is known also as chronic or interstitial pneumonia, cirrhosis, or fibroid induration of the lungs, and is usually accompanied by dilatation of the bronchi. It is generally of one side, protracted in its course, and is characterized by a change from the normal pulmonary tissue to the excessive formation of connective tissue, and is often associated with or followed by tuberculosis. It is held by some that fibroid phthisis begins as a tuberculosis in all instances. It is certainly proved that many inflammations of serous membranes aforetime ascribed to exposure to cold are really tubercular. This is especially true of the pleura. The affection is rare in children unless it be after measles or whooping-cough, they usually exhibiting a more active process during an inflammation, with a larger power of complete repair. When seen in adults, the origin may frequently be traced to childhood. A simple bronchitis may set up changes which result in an overgrowth of the interstitial connective tissue. (See Chronic Bronchitis.) It is exceedingly uncommon in children to find an abundant connective-tissue growth the result of an ulcerating tubercular process in the lung, although it is possible. The usual origin of fibroid phthisis in children is in the course of pneumonia and bronchopneumonia, especially the latter. Bronchiectasis, a cylindric dilatation of the bronchi, is also frequently present, due to excessive coughing. An entire lobe may be affected, but usually only a portion of a lobe or areas in the lung or the walls of the bronchi may be thickened.

**Pathology.**—The changes in the lung are usually upon one side

and in the lower lobes. The parenchyma of the lung appears destroyed and replaced by connective tissue. There is usually peribronchitic thickening and also bronchial dilatation, producing cavities of varying sizes. Cavities may also be formed through ulcerative change, particularly where secretions are retained and become decomposed. Tubercle bacilli, active or latent, may be found in the tissues or secretions or locked up in the connective tissue. The lung usually suffers some diminution in size, which is shown upon inspection. The pleura is generally affected, and adhesions are liable to be particularly dense. The sound lung exhibits compensatory hypertrophy and often emphysema. The heart rarely escapes displacement, and the cavities suffer change because of the increased resistance in the pulmonary circulation. A common result of pleural inflammation is adhesive pericarditis; this may be found here, as well as a general venous stasis.

**Symptoms.**—There are always in chronic bronchopneumonia cough and expectoration, the latter appearing in large amounts at long intervals. When cavities exist, the sputum may separate into three layers of froth, serum, and pus; it is often of a most offensive odor. If tubercle bacilli be found, they may betoken a recent infection from outside or a manifestation of latency. Elastic fibers will be shown if ulceration is present. Hemoptysis occurs, usually of moderate amount; but if a fair-sized vessel be affected, danger results, or possibly death. Even in favorable cases dyspnea arises upon exertion, or even, in worse ones, while the patient is at rest. Fever and its effects are absent or occur only intermittently. Should it arise, it is a symptom of some secondary complication. Nutrition suffers little, and the general health is fair. That infallible sign of chronicity, clubbing of the fingers and toes, is more constantly seen in interstitial pneumonia than in any other condition, except, perhaps, congenital heart affections. The physical signs are similar to those of chronic tuberculosis. Certain signs are, however, distinctive of this malady. The respiratory murmur is much impaired unless there is bronchiectasis, when the breathing is distinctly bronchial. The two sides of the chest show a notable difference in shape, mobility, and size, especially when pleural changes are extensive. Retraction is more common if the apex is affected. Exploration of the chest may reveal areas of consolidation, retained secretion, or vomicae; and in this malady more often at the base than at the apex. According to the shrinkage or change in the shape of the chest, or where in the pleura or pericardium adhesions have taken place, there are seen changes from the normal in the cardiac

impulse. Other changes the outcome of altered relationships between the lungs and the heart may be manifest, but are sometimes very obscure. If the compensatory hypertrophy of the right ventricle suffers, we have evidences of venous stasis, pulsation of the jugulars, edema, enlarged liver, cyanosis, and the like. The course of fibroid phthisis is essentially chronic, and, on the whole, progressive, although there may be periods of fair health, and yet death may occur from intercurrent disease.

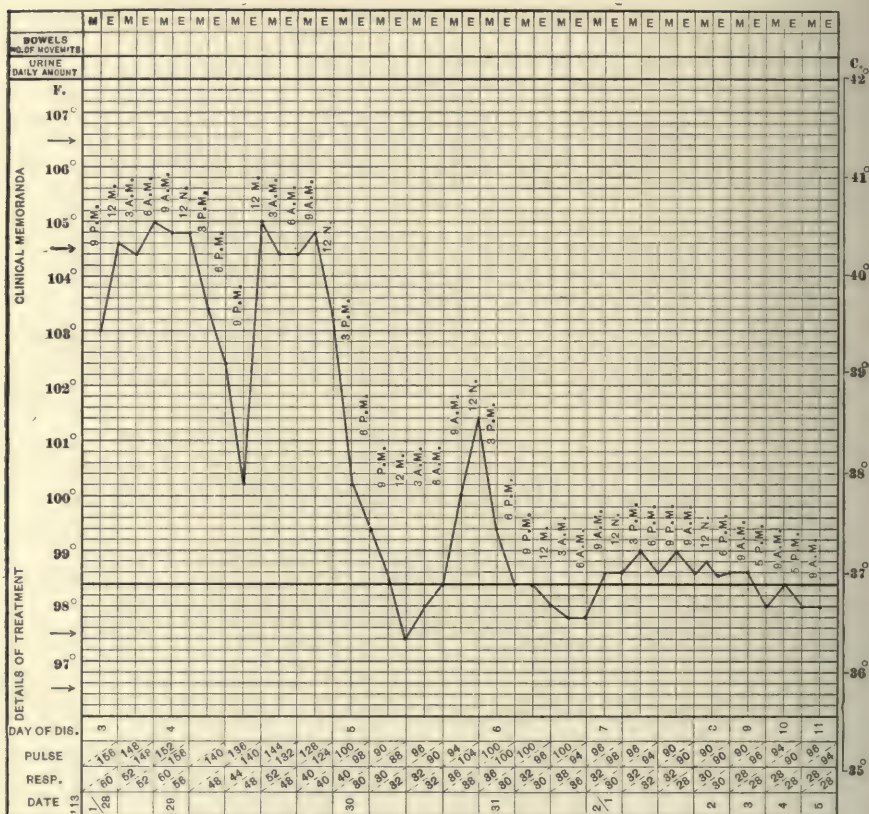
The **diagnosis** should not be difficult if a history can be obtained. Chronic tuberculosis is excluded on account of excellent general nutrition, the absence of fever, and the mode of onset. In fibroid phthisis there is a history of chronic cough and expectoration, with repeated blood-spittings, physical signs of lung destruction (generally of one side and often with the formation of cavities), or signs of shrinkage and hardening, and most often the process is an apical rather than a basal one. The heart exhibits dilatation soon or late, with dilated right ventricle. The disorder may be confounded with chronic pleurisy, in which there is also much contraction of the side. In cancer of the lung or pleura the thoracic physical signs are similar, but the course and duration are quite different.

**Prognosis.**—A sufferer from chronic bronchopneumonia may live a great many years, especially if the individual is in comfortable circumstances and can or will be reasonably careful. The danger is from intercurrent diseases. If the process is not too prolonged and the destruction of tissue too great, recovery may take place.

**Treatment.**—Nothing can be done to repair the damage to the parenchyma of the lung. Much, however, can be done to place the patient in good health and keep him there. In the accomplishment of this a judiciously regulated outdoor life, attention to the diet, and, above all, to the skin, will do much to lengthen the sufferer's days. Respiratory gymnastics are capable of doing a good deal in improving the condition of the lung, especially suitable exercises in a wholesome atmosphere. High altitudes are theoretically contraindicated, because of the deficient respiratory capacity. This must be determined in each particular case, estimating the collateral conditions, whether a dry or a moist climate be the best. Expectorants are to be avoided as a routine measure, but must be prompt and efficient when needed. Potassium iodid is useful in certain stages of bronchitis, and creasote or guaiacol will at times serve a useful end. Cod-liver oil is the stand-by always, and other nutrient tonics—malt, hypophosphites, etc.—are frequently of use.

## CROUPOUS PNEUMONIA

Croupous pneumonia, or lobar pneumonia, is a specific inflammatory disease of the lungs due to infection by the bacillus lanceolatus (pneumococcus) accompanied by exudation into the vesicular structure, with subsequent consolidation. Clinically, it is an acute, self-limited disease, manifested by high fever, hypernea, cough, and rusty sputum,



of measles, whooping-cough, influenza, typhoid fever, and tuberculosis, but in children under three years of age this is most unusual. Exposure to cold is the predisposing cause and depressing conditions are contributory, but the exciting cause is the bacillus lanceolatus (*diplococcus pneumoniae*) of Fraenkel. This microbe holds also close relations with empyema, middle-ear disease, peri- and endocarditis, arthritis and peritonitis. Among other bacteria causing pneumonia may be mentioned the bacillus pneumoniae (Friedländer) as well as the streptococcus and staphylococcus pyogenes. No doubt other bacteria are sometimes responsible for croupous inflammations, such as the bacillus typhi abdominalis. There is reason to believe that croupous pneumonia is contagious. We are lead to accept the modern view that the infection is a systemic or blood disease, that may evidence itself locally in lung, ear, pleura, pericardium, joints or peritoneum.

**Pathology.**—The lesions of lobar pneumonia occurring in children are much the same as those of the adult, being an acute exudative inflammation extending throughout the whole of one lobe or the major part of one lung or limited portions of both lungs. The stages are those of congestion, red hepatization, gray hepatization, and resolution, progressively, just as in the adult.

During the first stage, or the stage of engorgement, the lung tissue is congested. It is of a deep-red color, firmer to the touch than normally. On section a frothy liquid exudes, made up of serum and blood. The lung still crepitates, and a cut portion will float. The alveoli contain fibrin, leukocytes, red cells, and detached epithelium. The capillaries of the air vesicles are dilated and tortuous, and the alveolar epithelium is swollen. The first stage lasts a few hours or several days. When the output of inflammatory products has reached its height, these, collected within the alveoli and terminal bronchi, increase the size of the lung as well as its density; hence the stage of red hepatization. The lung is of dark-red color, solid, airless, and firm. It is easily friable, cuts like liver, and sinks in water. This stage occurs earlier in children than in adults.

Microscopically studied, the alveoli are observed to be filled by threads of coagulated fibrin, in the meshes of which are seen red blood-corpuscles, polynuclear leukocytes, and alveolar epithelium. The alveolar walls are infiltrated, and leukocytes are seen in the interlobular tissue. Thereupon follows a period in which the exudate becomes changed in color from red to a mottled gray; this is called *gray hepatization*, and is a process of degeneration and softening. The air-cells are filled with leukocytes. The fibrinous network and the red

blood-corpuscles have disappeared from the alveoli. Finally comes its last stage; in this the exudate is softened. Disintegration and degeneration of the cell elements continue until they are rendered soft and capable of absorption. The lymphatics take up and remove these products, and this is called *resolution*. During the course of an ordinary lobar pneumonia no permanent change takes place in the lung structure itself; consequently recovery occurs by resorption and expectoration of the exudate.

In children lobar pneumonia is frequently bilateral; the lobe most frequently affected is the lower one of the left lung, and pneumonia of the apex is quite prevalent among children, though rare in adults. A bronchitis may accompany the process in a fair proportion of cases. A plastic form of pleurisy occurs where the consolidation reaches the pleura; more rarely an effusion takes place and empyema may result. Pleurisy of a mild sort is frequently present yet unrecognized; it is less often a complication in lobar pneumonia than in bronchopneumonia.

**Symptoms.**—Lobar pneumonia, being a primary process, usually manifests itself abruptly, with few or no prodromes. The seriousness of the malady is promptly defined by the onset with vomiting or convulsions, or both, usually a certain amount of rigor or chilliness, with a pronounced rise of temperature. Convulsions are not seen, however, as commonly as some would lead us to believe. Personally, we do not think they occur, unless there is an underlying convulsive tendency. The clearly marked chill, so common in adults, is rarely seen; instead of this the nervous system sometimes loses its balance (convulsions) or a mild delirium often appears, or the stomach rejects its contents. The temperature rises rapidly, and inside of a day may reach 104° or 105° F., continuing with slight daily remissions and declining by rapid crisis from the third to the ninth day. It often then falls below the normal. The pulse is full and bounding, increasing in rapidity as the temperature rises, but respiration is accelerated in even greater proportion than either, so that the pulse-respiration ratio of one breath to two pulse-beats is pathognomonic in this disease. The respirations are usually panting—like those of a dog. Pain is usually present, referred vaguely to the chest or abdomen; dyspnea may become a most urgent symptom. A short, dry cough may appear early, which sometimes seems to give rise to pain. The cough, however, may not come on until later, and changes its character as the disease progresses. There is rarely any expectoration before the seventh or eighth year, and then it may exhibit the rusty character common in adults. The face is often flushed, or circumscribed spots of

redness appear on the cheek; the eyes are bright and the facial expression is anxious; the alæ of the nose are dilated, showing increased inspiratory effort. In the milder cases there are restlessness and irritability; in severer ones there is apathy, at times complete. Appetite is usually lost, and sometimes marked digestive disturbances persist throughout the disease. In certain severe cases the nervous symptoms are prominent throughout, usually in proportion to the height of the fever, and death may occur in a convulsion before even the characteristic physical signs appear. If the nervous phenomena appear at first only, they are of little gravity; if they arise later, they are of gloomy portent. In intensely severe cases the dyspnea and cyanosis develop markedly, the respiration is shallower, the pulse more rapid and weak, the child becomes stuporous, and death occurs quietly or with motor excitements.

Pseudo-crises are far more common than in adults, and very often the patient appears more ill after a pseudo-crisis than at any previous period of his illness. The true crisis usually appears earlier (fifth to seventh day) than in adults; but one must be prepared to meet the following aberrant terminations of the disease: 1. Fall by lysis. 2. Wandering pneumonia, when two, three or more lobes are successively attacked. 3. Delayed resolution. These terminations are more or less peculiar to childhood, and should not catch the medical attendant off his guard.

The physical signs in lobar pneumonia in children are practically the same that we find in adult life. The thin chest walls of the infant and child render the lungs more accessible to examination though, and in general the diagnosis is easier to make in the young. This statement needs certain qualifications, however: It is highly probable that central pneumonias occur, though they are certainly rare, and in such cases, careful examination may fail to reveal the pneumonic area for several days. Again, we may see patients whose symptoms are most atypical, and it is only upon the physical finds that one may depend (see below). The principal signs are as follows:

**Inspection.**—Play of alæ nasi; accentuation of the nasolabial line; limitation in movement of the affected side; accentuation of abdominal respiration.

**Palpation.**—Increased fremitus over the affected lobe and possibly over the well side (compensatory). This may be deceptive, however. Rhonchial fremitus is not noted, as in bronchopneumonia, unless there be an accompanying bronchitis.

**Percussion.**—This should be very light as we have previously stated. Such light percussion will usually elicit hyperresonance over

the unaffected side, and over the unaffected lobe of the diseased lung. Over the involved lobe, there is impaired resonance and this is usually obtained over the whole lobe. Resistance is not increased to the extent that it is in pleural effusion.

**Auscultation.**—On the first day one may obtain weak vesicular respiration over the affected area; but after this there is usually bronchial or actual tubular breathing. Early one may hear crepitant rales, certainly more often than in adult patients. Then for days the case may be characterized by its absence of rales. Finally with resolution, large moist rales (*redux crepitus*) are heard. The voice sounds are usually modified, bronchophony being a constant find. There may be great variations in all symptoms; in some cases the cough is absent until many days elapse; the cough and physical signs sometimes remain for several days after the temperature has dropped. The presence of pleuritic exudate produces a dullness, with only a muffling of the breath-sounds. There are, moreover, certain varieties of lobar pneumonia which have received special names and vary considerably in their symptomatology, in some cases strongly simulating meningitis with hyperpyrexia, convulsions, delirium, or coma, and yet without cough (*meningitic pneumonia*.) These cases are liable to arise among debilitated children, and the pneumonia is often of the apex. Abdominal pneumonia is a name given to those cases marked by digestive disturbance, vomiting, diarrhea, and abdominal pain. These may be so severe as to simulate peritonitis (*appendicular variety*). The pneumonia in these cases is only to be discovered by careful search. Wandering pneumonia is very like bronchial pneumonia in certain of its symptoms, but not so entirely similar as to fail of a differentiation.

Leukocytosis is an almost invariable accompaniment of croupous pneumonia. (See Diseases of the Blood.) Early the blood pressure is always elevated.

The commonest complication of croupous pneumonia is middle ear disease, particularly in early life (Morse), and ranking as a close second we have pleurisy. No doubt many mild pleuritides escape notice. The effusion into the pleural sac may be plastic but is usually purulent, especially when following measles and scarlet and typhoid fevers. Pericarditis may complicate the pleurisy or occur alone. The meningeal form, or, better termed, the cerebral form, is very alarming, but rarely does this indicate a true meningitis (*meningismus* or *toxic meningitis*). Disturbance of the kidneys occasionally arises. Abscess and gangrene are rare sequels.

**Diagnosis.**—When seen in the fully developed form, a lobar pneumonia may be difficult to differentiate from a bronchial pneumonia. Wandering pneumonia with areas of fleeting consolidation is even more confusing. The abdominal type of pneumonia is readily overlooked, and here the respiration and pulse-rate must be carefully measured. Leukocytosis, as mentioned, is present in pneumonia; and often a urinary examination reveals a diminution in the chlorides. The suddenness of lobar pneumonia and its high fever, vomiting, and convulsions before the physical signs are manifest, together produce a marked similarity to the onset of scarlatina. Pleurisy with effusion is to be recognized by its movable character, absence of breath-sounds, râles and by changes of vocal resonance (egophony) and onset with pain.

In the diagnosis between the cerebral form of pneumonia and meningitis it is important to bear in mind that in the former we have rapid pulse and hurried respiration. In meningitis there is usually a slower pulse, with slow, irregular breathing. In lobar pneumonia usually a number of its own characteristic features will appear to distinguish it from the marked irregularities, slow, insidious onset, irregular temperature, pulse and respiration rates of bronchial pneumonia. The unilateral character of lobar pneumonia will mark its distinction from the bilateral manifestations usual in bronchial pneumonia.

**Prognosis.**—Bronchial pneumonia in children is a far graver disease than primary croupous pneumonia. In the secondary form of croupous pneumonia, however, it is more serious, and in the septic cases the mortality is high. Of evil omen, too, are pronounced cerebral symptoms, hyperpyrexia, and great dyspnea, but experience proves that even the severest cases recover surprisingly. A low leukocyte count may also be of bad prognostic omen. The mortality is about 5 to 10 per cent.—it is large under three years but very small from that age to ten years.

#### PLEUROPNEUMONIA

Pleuropneumonia is recognized as a separate variety of pneumonia accompanied by an excessive degree of pleurisy, along with exceptionally marked consolidation. The pleurisy in these cases usually arises simultaneously with the pneumonia, or it may follow. The cause is oftenest the pneumococcus. The form is a bronchopneumonia in two-thirds, a lobar pneumonia in one-third, of the cases. The left lung is affected in two cases out of three, the pleurisy being of both lungs, as a rule, in different degrees. Both surfaces of the pleura are

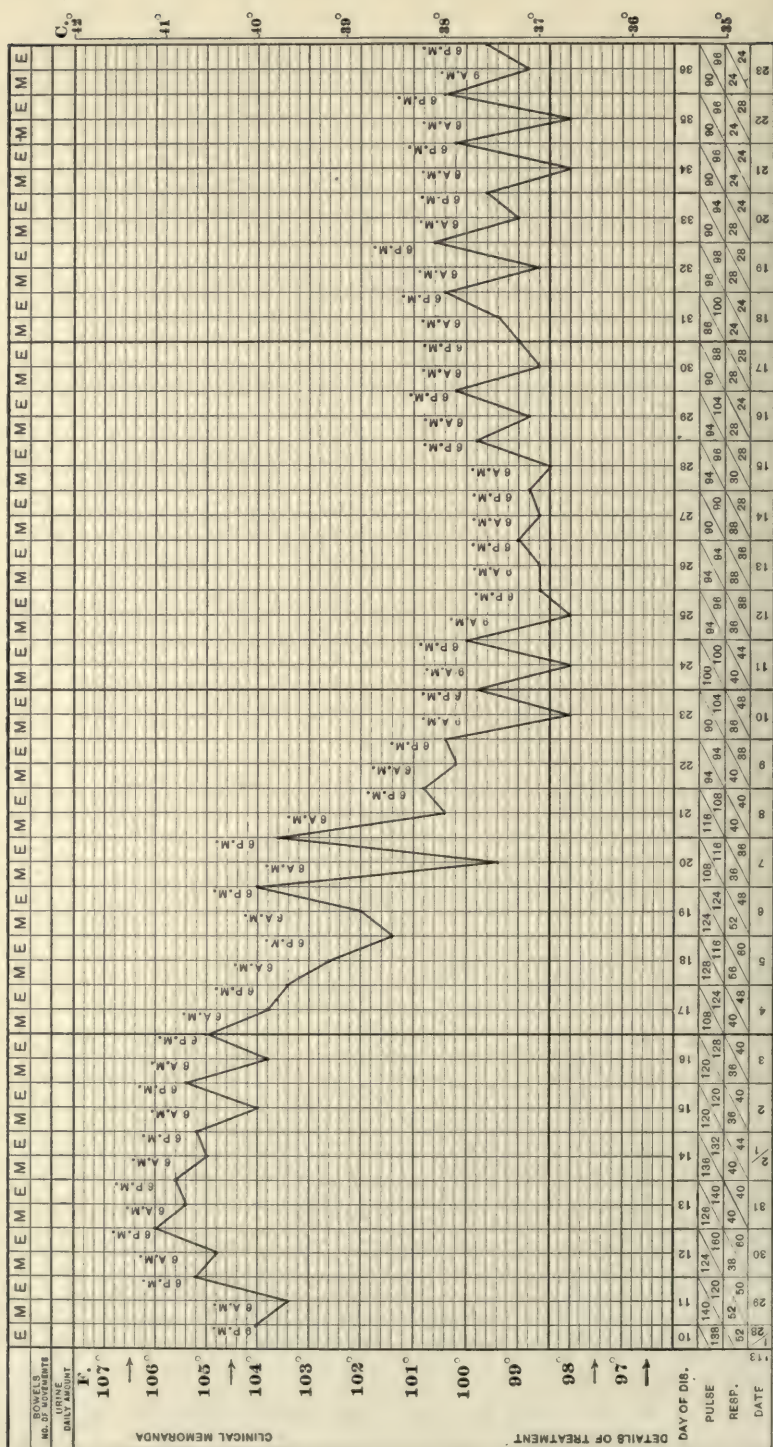


FIG. 57.—WANDERING PNEUMONIA, INVOLVING PRIMARILY THE LEFT UPPER LOBE; THEN THE RIGHT LOWER; THEN THE UPPER LOBE OF THE RIGHT LUNG. LATER ON HE HAD A DOUBLE OTITIS MEDIA. EVENTUAL RECOVERY AFTER TEMPORARY LOSS OF SPEECH.

found covered with greenish-yellow fibrin, glueing the opposite walls together, affecting also, probably, the pericardium and the diaphragm. The intensity of the inflammation is liable to cause a fatal result early in the course of the disease. When the chest is filled with pus, the condition is called empyema. The exudate may rarely be only serous. Absorption may take place, followed by adhesions, usually extensive. The symptoms differ little from those of a combined pneumonia and pleurisy and chiefly in the degree of severity of the constitutional symptoms, pain, temperature, and subsequent exhaustion. The auscultation sounds are exceedingly puzzling.

The **prognosis** is naturally bad; infants usually die in the acute stage.

The **diagnosis** from empyema or simple effusion is not so difficult if punctures are made. It is difficult to withdraw the fluids unless a pocket be accidentally punctured.

**HYPOSTATIC PNEUMONIA** cannot be readily diagnosed, but commonly accompanies fatal chronic or wasting disease, particularly marasmus in infants. The lesion postmortem is seen to be confined to a superficial strip along the posterior borders of both lungs, not involving the deeper structures, as in atelectasis. This should not be regarded as accounting for the death.

### THE TREATMENT OF PNEUMONIA

**Treatment.**—Prophylaxis is of prime importance. Such measures as we have emphasized in the treatment of coryza and bronchitis will serve to prevent many secondary pneumonias. When the pneumonic process is in full blast, prophylaxis may still serve to prevent complications. We believe that this statement possesses special significance in its relation to the most common complication—otitis media. The auditory canal should be packed with carbolic acid in glycerine, and the ears further protected from low temperatures by a woolen cap. One other preventive measure demands emphatic expression: The contagiousness of pneumonia is often demonstrated in hospital and private practice. Pneumonias complicating measles, diphtheria and epidemic influenza seem to possess this quality in high degree. On several occasions, we have treated two children, in the same family, with pneumonia and only recently, one of us was called into a family in which three of their five boys had fallen ill of this disease. Isolation is therefore a wise measure.

**Active Treatment.**—General measures: It goes without saying that the patient should be kept in bed during his disease, and for a week

or more after his crisis has occurred. He should be clothed in a single garment, so constructed as to be readily removed and replaced by another. A pajama suit of wool, or woolen underdrawers with feet, will meet these requirements. The bed clothing should be light in weight, but warm. It is well to employ both urinal and bedpan, unless the patient is much disturbed by their use. Hypostasis should be prevented by frequent changes in the patient's position in bed.

**Fresh-air Treatment.**—Had Northrup done nothing else to place pediatrics on the high plane it has reached in America, he would deserve everlasting credit for his forceful teaching on this subject. In our own minds, he shall be likened always to the great Willis, who “fed fevers.”

The oxygen tank is seldom needed, when cool or cold fresh air gains free access to the pneumonia patient. In croupous pneumonia, with high temperature, the inspired air can scarcely be too cold, and “balcony” or “roof” treatment may be employed. Blood pressure is raised; respiration is deepened and the rate slowed; the heart action becomes slower, and delirium is often calmed. As we have stated, the body surface must be well protected meanwhile, and the sensitive ears sedulously guarded. In the presence of broncho-pneumonia, particularly in young infants, and when the bodily temperature is low, the fresh-air treatment must be modified considerably. In the summer period, electric fans have served us well, and in hyperpyrexia accompanying croupous pneumonia, we have permitted a fan to play over a tub of ice placed beside the patient's bed.

**Diet.**—This must be varied according to the age of the patient, and the nature of his case. In general, we prefer to feed at three-hour intervals, just as we prefer to employ medicinal and local measures at three-hour intervals. We say to the nurse or mother: “Everything that is to be done for the patient, must be done in from 20 minutes to one-half hour. The sufferer will thus be able to rest for from two and one-half hours to two hours and 40 minutes in every three hours.” To older children, we give milk, with or without malted milk, and fruit juices with raw eggs. A raw egg shaken with orange or lemon juice and cracked ice usually proves most acceptable to the patient. Water should be given freely and should be administered cold. Infants, and particularly infants ill with broncho-pneumonia, should receive peptonized milk. They are thus saved the expenditure of energy required in digestion, and are also safeguarded against the gastro-intestinal disturbances that may turn the tide against them. In such cases, too, it may be necessary to give food more frequently and in smaller amounts,

even though, theoretically, such frequent disturbances do not appeal to us.

*Local measures, as applied to the chest*—many of these procedures have been employed by us; but none has proved quite so valuable in our hands as the mustard paste advocated by Holt. Heubner, too, is an advocate of mustard as a counterirritant, though his technic of employment differs from that of Holt. The paste is made by mixing 1 part of yellow mustard with 6, 5 or 4 parts of flour, and some warm water. It is applied front and back between layers of linen or cheese-cloth. The length of application is determined by the resulting action on the skin. The skin is usually well reddened in from five to ten minutes. We then rub the reddened area with a 5 per cent. or 10 per cent. ointment of guiacol in lanoline. A light cotton jacket, made in two parts (front and back) is then reapplied. We are very specific in our directions to the nurse. They are: "Take off the front of the jacket, and apply the front of the paste. Turn the patient over, and take off the back of the jacket and put the paste on the back. When marked redness of the skin results, take the paste from the front of the thorax, rub briskly with the ointment and replace the front of the jacket. Turn the patient over, and repeat the same procedure." Exposure is thus avoided.

Usually the warm application has such a tranquillizing effect, that we feed the patient while the paste is on. As the pneumonic process subsides, the patient's skin cannot stand the counterirritation. This we have considered one of the strongest arguments in favor of counterirritation. We treated one baby with broncho-pneumonia in a distant city. For nearly six weeks he had three-hour applications of mustard paste. Before the time for a given application, the redness of the skin induced by a previous treatment had always disappeared. But when his pneumonia began to resolve, we had to lengthen more and more the intervals between treatments.

The Priessnitz compress has also yielded good results in many hands. Ice, locally applied, does not appeal to us, and we have come to view cold applications to the chest as depressing and dangerous to babies and young children.

**Stimulants.**—We admit freely that many cases of croupous pneumonia will proceed to favorable terminations without any other measures than those just outlined. But on the other hand, we are not in sympathy with therapeutic nihilism in the treatment of this disease. In our own experience, most of these patients need stimulation, and most of them are better off when they get it. Alcohol, usually in

the form of brandy or good whisky, serves several useful purposes. If food is well taken, the stimulant may be given in the food. It is rapidly burned up in the system, yields energy promptly, and so spares nitrogenous waste. It often has a tranquillizing effect upon the disturbed cerebrum, and it serves to send more blood to the body surface. In proper dosage, it is rarely noted on the breaths of pneumonic patients. Under a year of age, babies may be given from 10 to 20 drops of spirits; in the second year of life double these amounts. Children may receive one or two drams of brandy or whisky. Champagne is of value in cases presenting the so-called typhoid state. It may be given more frequently than whisky or brandy. Aromatic spirits of ammonia we give in a routine manner, following several authorities. Usually, it is given before food. Despite much argument against this mode of administration, we give strychnine to most of our patients. We find that when it is administered, the children take more food, their hearts seem to beat the strain beats, they have less depression late in the disease, and they present fewer alarming symptoms in the early morning hours. Digitalis, too, is useful. It slows the pulse even during the height of the disease. Cardiac energy is probably thus conserved. If it does not slow the pulse in two or three days, its employment should cease. It is our custom to give strychnine and digitalis every six hours, and to avoid too many medicines, we give them together in a mixture. This may seem unscientific; but accurate dosage is secured by means of a minim glass measure. Thus, to a baby of two years, we would prescribe:

R. Strychnin. nitrat.....	gr. $\frac{1}{2}$
Tr. digitalis.....	ml xviii
Glycerin.....	fl. $\overline{5}$ ij
Aq. cinnamom.....	q. sad. ft. fl. $\overline{3}$ ij
M. et solve	
SIG.—fl. $\overline{3}$ i (not a teaspoonful) every six hours.	

In the presence of serious cardiac depression, late in the disease, strychnine may be employed in larger doses, may be given hypodermatically and at more frequent intervals. Other stimulants of value are camphor in sterilized olive oil, glonoin, caffeine-sodium-benzoate and atropine. We have seen many lives saved by the use of camphor in olive oil. Unduly protracted cases, unduly toxic or septic cases, cases with pronounced nervous symptoms, and patients with pronounced weakness of the muscular sound of the heart, seem to respond better to the hypodermatic employment of camphor and olive oil, than to any other remedy we have employed. In the presence of pulmonary edema or

in cases which present unduly low temperatures following crises, atropine is a remedy of signal value.

**Expectorants.**—In most cases of croupous pneumonia, expectorants are not indicated. In cases with delayed resolution, however, ammonium iodide and creosotal are useful expectorants. In broncho-pneumonia, we use expectorants, though usually the aromatic spirits of ammonia is all-sufficient for this purpose. Ammonium carbonate is also a useful drug, as we have indicated in our treatment of bronchitis. The employment of the guaiacol ointment indicated, means that we are administering creosote to these patients.

**The Reduction of Fever.**—In most cases of croupous pneumonia, no antipyretic measures need be employed. In broncho-pneumonia, the fever is of a remittent type, and we quite agree with Holt, that any temperature between  $101^{\circ}$  and  $104^{\circ}$  should be viewed with little concern. In cases presenting hyperpyrexia, and in unduly protracted cases (wandering pneumonia), the fever itself may constitute a menace. Under such conditions, cold fresh air may prove a veritable boon.

The ice-cap is useful in older children; but may be hard to keep in place in infants. Hydrotherapeutic measures are of most value: Babies are often helped by hot baths or by hot mustard baths. Older children may be given a cool sponge, or a half-strength alcohol rub. Cold baths and cold packs we do not employ. Colonic lavage with cool ( $80^{\circ}$ – $90^{\circ}$ ) normal saline solution is an antipyretic measure of importance.

**Sedatives.**—We fear to use opium and its alkaloids in the pneumonias of early life, though occasionally we give Dover's powder in small doses. Acetphenetidin in grain doses sometimes acts well in the control of extreme restlessness. In more extreme cases we give ammonium bromide and chloralamide in combination. As a rule, however, the open-air treatment, the mustard paste and the hydrotherapeutic measures mentioned will serve to make the patient comfortable, and he will rest.

**Treatment of Complications and Sequelæ.**—If an ear drum membrane loses its luster, inflames and bulges, it should be treated with a warm solution of carbolic acid in glycerine, and if prompt improvement does not result, paracentesis should be performed. Abdominal distention (tympanites) should be viewed as a dangerous complication. Turpentine by mouth, and turpentine or asafœtidæ in an enema, seem to yield us the best results. Empyema must be dealt with surgically, and so must a true pneumococcic peritonitis. Meningitis we strive to prevent by attention to both ears and nose; but should it occur,

no treatment will prove of avail. Pericarditis, too, is likely to be purulent. If so it should be dealt with surgically. Affected joints should be mobilized in a removable plaster-of-Paris dressing, and if fluid forms despite this enforced immobility, aspiration should be performed. If the fluid is purulent, surgical treatment should not be delayed.

**Convalescence.**—As in scarlet fever and diphtheria, the patient's heart muscle should be safeguarded from strain. He should never get up in less than a week. Nutritious food is demanded. Tonics, like arsenic, iron and cod-liver oil are of great service. The tincture of nux vomica in port wine or grape juice, will stimulate appetite amazingly. Should the family's means permit, the patient should be sent south for the remainder of the winter season.

**Serum Therapy.**—That this treatment will assume much importance in the near future, we have little doubt; but up to the present time, the results of serum therapy seem inconclusive. Sometimes there is a sudden fall in temperature and a diminution in leukocytosis, both promptly following the injection. We have had no personal experiences with the bacterins.

GANGRENE OF THE LUNG occasionally occurs in feeble children of poor nutrition, usually under three years of age, and following the course of depressing diseases, particularly bronchopneumonia and measles. One of us observed it in a boy of ten years convalescent from typhoid fever. The immediate cause is the admission of saprophytic organisms to devitalized lung tissue.

The distinctive symptoms are the gangrenous odor of the breath and the expectoration of fragments of decomposed lung tissue. The sputum separates into the three characteristic layers. Death, however, is liable to occur before these evidences are clear, and the diagnosis is usually made from postmortem findings.

**PULMONARY COLLAPSE (ACQUIRED ATELECTASIS).**—A condition of collapse may come upon areas of a competent lung during the progress of pulmonary disease or owing to causes which profoundly disturb the lung circulation or the pressure of air in the lungs.

This may arise from compression or obstruction. Collapse due to compression commonly accompanies pleuritic effusion or pneumothorax, pericardial effusion, cardiac enlargement, deformities of the chest, and thoracic or mediastinal new growths. This may be partial or complete, and becomes less remediable the longer it remains, especially if there exist dense pleuritic adhesions, which last may be the chief barrier to reexpansion. Collapse from obstruction is due to two factors—blocking of the bronchial tubes, great or small, and in-

competent respiratory vigor. Holt says that this first factor has been greatly exaggerated. If the lumen is narrowed, the stenosis is most liable to result in emphysema. When a bronchus is obstructed from any cause, usually from a foreign body or from external pressure preventing the entrance of air, that portion of lung beyond this point becomes slowly collapsed; if a primary bronchus, the whole lung; if a lobar division, the whole lobe; if a bronchiole, a small contiguous area. The collapsed portion becomes depressed below the normal surface, is of dark-red color, highly vascular, and resembles pneumonia—in which it may result. Holt, also from special observation, declares that the development of emphysema is much more likely to result from stenosis, due to bronchitis of the smaller tubes, etc., rather than from atelectasis.

Collapse of areas of the lung may come on slowly and generally in feeble infants, rachitic and otherwise depressed, accompanied by bronchitis resembling congenital atelectasis, with much the same phenomena. The symptoms are rapid respiration, dyspnea on inspiration, sinking in of the chest-walls, cyanosis, and impaired peripheral circulation. Congenital atelectasis has received attention.

Hernia of the lung sometimes occurs, pouting out at each respiratory action. Should deformities of the chest-walls occur, respiratory gymnastics help more than anything else, and should be continued for months or years. The bronchial and mediastinal glands frequently become congested and inflamed in divers diseases of the thorax, though frequently are the result of protracted bronchial and nasal catarrh, or metastasis from other glandular disturbances, especially in rachitic or tubercular cases. The principal symptoms are those of pressure upon the tracheal veins, or nervous attacks of coughing with crowing inspirations, changed voice sounds, bronchial respiration, dullness over the sternum (upper part), and dullness about the interscapular region (Manubrial area, "stripe-dullness"). The control of this complication difficult, and consists of absorbents applied externally, mercurial ointment, or potassium iodid and iodine. Internally, arsenic, iron and other aids to nutrition should be used.

## PLEURISY

### Synonym.—PLEURITIS

We also speak of acute, subacute and chronic pleurisy or pleuritis. Again, the particular type of exudate leads to the use of the adjectives *fibrinous*, *sero-fibrinous*, *fibrino-purulent*, and *purulent* in the descrip-

tion of forms of pleuritis. Purulent pleuritis is commonly designated empyema, and in general, empyema may be viewed as the form of pleurisy peculiar to infancy and early childhood. Netter states that from one-third to one-half of all pleuritides in childhood are purulent; while this is true of but one-fifteenth to one-sixteenth of the pleural inflammations in adult life.

**Etiology.** *Age.*—Pleurisy may occur at any age. Like peritonitis, it may be seen in the new-born. Our youngest case was observed in an infant five weeks old. Netter finds that two-thirds of all empyemata occur in the first five years of life; one-fourth from the sixth to the tenth years, and one-tenth from the eleventh to the fifteenth years. (Summary of 642 cases.) Of 145 cases, cited by the same authority, 46 occurred in the first year; 30 in the second; 22 in the third, and 16 in the fourth. It may be stated, therefore, that the younger the patient, the more likely is his pleurisy to prove purulent. Beyond the sixth or seventh year, the sero-fibrinous form of exudate becomes the most common.

*Sex.*—Boys are more often affected than girls, probably because pneumonia is more common in males.

*Primary and Secondary Forms.*—Inflammation of the pleura may be primary, but it is more frequently secondary to some other disease. Croupous pneumonia is the disease that most often precedes the pleuritis. Broncho-pneumonia is not infrequently complicated or followed by purulent pleuritis (empyema). Other preceding affections of note are pulmonary gangrene, pulmonary tuberculosis (usually responsible for fibrinous or sero-fibrinous pleuritis), caseous bronchial glands, caries of the vertebræ, pericarditis, appendicitis, scarlet fever, tonsillitis, diphtheria, grip, measles, whooping cough, typhoid fever, erysipelas, variola, rheumatism, intestinal or general sepsis, osteomyelitis, peritonitis, nephritis and syphilis.

**Bacteriology.**—Practically all inflammations of the pleura are bacterial in origin, though it is not always possible to detect the offending organism. Effusions that appear free from bacteria are usually tuberculous in nature. Schkarin found germs in sero-fibrinous exudates—most often pneumococci. All investigators who have devoted attention to the subject of empyema recognize three principal varieties of purulent exudates—those dependent upon the pneumococcus; those caused by streptococci or staphylococci, and those of tuberculous origin. Much more rarely, the colon bacillus, pyocyaneus or other organism may be found. This recognition of three principal groups of empyemata is important not only from the standpoint of causation,

but also from diagnostic, prognostic and therapeutic aspects. In childhood, the pneumococcus is the most frequent offender, while in adult life the ordinary pyogenococci more often play in etiologic rôles.

Netter's comparative figures are interesting:

	Pneumococci	Streptococci	Tubercle bacilli
Adults (154 cases).....	24.9 per cent.	41.2 per cent.	17.6 per cent.
Children (90 and 81 cases)	80.7-65.4 per cent.	13.3-19.7 per cent.	5.5-7.4 per cent.

Koplik found pneumococci in 60 per cent. of cases; streptococci in 15 per cent., staphylococci in 9 per cent., and tubercle bacilli in 7 per cent. Blaker found pneumococci in 94 per cent. of cases. Hughes study gave results closely in accord with those of Koplik. In one of our cases, Dr. Evans recovered an unidentified monococcus.<sup>1</sup>

**Pathology.**—Pathologically, we may recognize plastic pleuritis, sero-fibrinous pleuritis, empyema and pyopneumothorax. In practically all cases of pneumonia or tuberculosis of the lungs, a plastic exudate appears over the contiguous pleura. The membrane loses its luster, becomes thickened and presents a hyperemic surface. The deposit of fibrin appears in the form of flakes or larger deposits of pseudo-membrane. In apical tuberculosis, the two pleural surfaces overlying the tuberculous process become thickened and permanently adherent. Such adhesions are more commonly found posteriorly (Hektoen and Riesman). In certain cases of tuberculosis, particularly in infants and young children, the whole pleural surface may present such changes, and eventually the thickened pulmonary and costal pleuræ become bound to one another, thus obliterating the whole serous sac. Miliary or larger tubercles may be in evidence in the resulting thickened mass. Barring the distinctly localized forms, however, few cases of fibrinous pleuritis remain of this plastic character. Rather they tend to pass over into a sero-fibrinous stage, and as in adult life, so in later childhood, this becomes the most common form of pleural inflammation. The serum is readily distinguished from the fluid of a mere transudate. It represents a distinct exudate. Its color is ordinarily yellowish green, and its specific gravity usually ranges above 1025. It contains few organized bodies—leukocytes, a few erythrocytes, distorted endothelial

<sup>1</sup>Most of these data are derived from the article of E. Feer of Basle in "Pfaundler and Schlossmann's" System.

cells and shreds of fibrin. In a recent case, studied with Dr. Knipe, a large coagulum of fibrin quickly formed in the aspirated fluid, and in this fluid Dr. Duncan found evidence of a marked endotheliosis. In the most dependent portions of the pleural sac, curds of a "white or creamy appearance" and a "buttery consistency" are found (Hektoen and Riesman). The lung floats upward in the large effusion, unless it is bound in place by adhesions. Resorption of the fluid may be slow or rapid. With this absorption of the exudate, permanent white patches of connective tissue or larger white callous thickenings are noted on both pleural surfaces. Curd-like or more extensive adhesions also form between the two surfaces. If the lung is free to move, the resulting fibrous bands of adhesions may be stretched more and more until they come to resemble chorda tendineæ (Hektoen and Riesman). As already mentioned, extensive adhesions may obliterate the pleural cavity, or they may bind the lobes of the lung firmly together or actually extend into the interlobular septa. Such dense and general adhesions usually result when there is extensive tuberculous disease of the lung. In a large proportion of cases, the sero-fibrinous exudate is apparently free from bacteria, and in most of these cases the pleuritis is tuberculous in origin. This form of pleuritis, however, may accompany pneumonia, pericarditis, rheumatism, typhoid fever or other infectious disease. Empyema we have defined as the pleuritis most likely to occur in infancy and early childhood. We have mentioned also its principal varieties. Rarely a sero-fibrinous pleuritis may become purulent, particularly in a tuberculous subject or when infection occurs from the outside (as in aspiration); but usually an empyema is a purulent process from the first. In the pneumococcic variety, the pus is generally thick, creamy and greenish yellow in appearance. It also contains flocculi or larger masses of fibrin. Sometimes great layers of this fibrinous exudate may be superposed upon one another, making successful aspiration or drainage difficult. Interlobar empyema is far more common than is usually supposed. We have seen at least seven such cases. T. Turner Thomas believes that pleural adhesions play important rôles in the pathology and treatment of empyema, and in some cases of interlobar empyema (two of them seen with him), one of us has had ample naked eye evidence of their existence. We are convinced that most of the so-called encysted empyemata and most abscesses of the lung (barring those resulting from foreign bodies) are in reality interlobar empyemata.

When streptococci or staphylococci are responsible for the purulent pleuritis, the pus is much thinner in character, and less fibrin is found.

Tuberculous empyemata present a pus still thinner in character, and in such exudates bacteria may appear absent. Laboratory predigestion of the fibrin in the exudate and subsequent centrifugation may aid in the detection of tubercle bacilli; or inoculation of guinea-pigs may reveal the tuberculous nature of the process. In tuberculous exudates, too, the microscope may reveal a preponderance of lymphocytes.

An empyema may rupture externally (empyema necessitatis), may empty itself into a bronchus, may communicate with a pulmonary vomica (producing a pyopneumothorax), may rupture into the pericardium or may burrow to regions below the diaphragm. Spontaneous evacuation through a bronchus, we have seen in at least three instances. In one of our cases, to be described in the chapter on brain abscess, an empyema burrowed through the diaphragm and communicated by a fistulous tract with the stomach.

**Symptomatology and Clinical Course.**—Of plastic pleuritis and sero-fibrinous pleuritis, we shall have little to say. The conditions under which such pleuritides occur have been sufficiently detailed in our consideration of their etiology. The symptoms and physical signs of these conditions do not differ greatly from the subjective and objective phenomena of the same affections as observed in adults. When a lung is extensively diseased in tuberculosis, the possible existence of a dense adhesive pleuritis should be borne in mind. In such cases, we have made repeated exploratory punctures, all resulting in dry-taps. The condition may be suspected when, despite the apparent presence of a large pleural effusion, the heart and liver are not displaced. Another peculiarity of pleural effusions in childhood is that tactile fremitus may be increased even over a large pleural exudate. Again, as many authorities have stated, tubular breathing may be heard over a pleura filled with fluid. Probably the “wooden resistance” offered to the pleximeter in percussion is the most valuable single evidence of pleural effusion. Mensuration is also helpful.

When the antecedents of empyema are duly considered, the careful medical attendant should scarcely be caught napping. When the pneumonic crisis has occurred and the child continues to have some fever, or when the course of the pneumonia itself seems unduly prolonged, the attending physician should always bear in mind the possibilities of otitis media, empyema and unresolved pneumonia.

Let us picture a fairly typical case: A child three or four years old has a pneumonia. After five days or more, his temperature falls within a few hours to the normal point. The following day, the doctor is disappointed to find that his temperature has again risen to 101° or

more. Possibly the patient complains of pain, though often this is referred to the abdomen. If the pain is severe, it is evidenced graphically when the little subject coughs or cries. Day by day, the temperature curve shows more or less irregular remissions. Very soon the phenomenon of sweating appears. The patient's appetite is poor, his tongue becomes furred, there may be a sweetish odor to the breath, nutrition suffers and in a few days the child may present a cachectic or septic appearance. The pulse rate is usually disproportionately high and so is the respiration rate. The respirations may become labored, and slight cyanosis may appear. An examination of the blood will reveal the existence of leukocytosis, with the polymorphonuclear cells in excess. Untreated, the subsequent history of the case will depend upon the character and virulence of the infection, and upon the resisting powers of the patient. Some children may carry such purulent collections for weeks or even months, while others evidence marked sepsis in a few days' time. Probably the symptomatology and physical signs may be best appreciated by comparing them to those of pneumonia and this we shall proceed to do in tabular form.

**Diagnosis.**—In the tabulated description (page 493) the principal symptoms and signs of pleural effusions have been accorded sufficient mention. By attention to the history and by an orderly elicitation of symptoms and physical signs, the medical attendant may usually arrive at a proper diagnosis. The condition from which empyema must be differentiated most often is *unresolved pneumonia*. The main points of distinction have been cited in the above table. Sometimes, however, the problem is complicated by the appearance of an empyema ere a pneumonia has resulted. Under such conditions, the exploring needle may yield the desired information. Otitis media should be suspected when pressure upon the tragus or mastoid process elicits tenderness, and the employment of the aural speculum will reveal a reddened or bulging drum, epithelium in the canal or a "sagging" posterior wall. Pulmonary abscess and gangrene of the lung both exhibit coarse râles and later signs of cavity formation. In the presence of the latter condition, the foul odor of the breath and the separation of the sputum into the three characteristic layers will furnish valuable diagnostic aids.

**Prognosis.**—This depends upon a number of factors, among which the following deserve consideration: 1. The age of the patient. 2. The type of invading organism. 3. The length of time that the empyema has existed. 4. The type of treatment pursued. 5. The presence or absence of other morbid states.

1. In general, the younger the patient, the more serious is the outlook. In very early infancy this age factor is accentuated because the empyema may represent but a phase of septic infection of the new-born.

2. Pneumococcic empyemata are most benign; tuberculous empyemata are more serious, and pus collections caused by streptococci and staphylococci offer the worst outlooks.

	EMPYEMA	PNEUMONIA
History .....	Of a pre-existent or existing pneumonia; of a former grip, measles, scarlet fever, some suppurative process, or tuberculosis.	Of a pre-existing illness with secondary broncho-pneumonia. Characteristic sudden onset with croupous pneumonia.
Symptomatology.....	More or less indefinite. Irregular fever, with unduly rapid pulse and respiration rates. Respirations are labored. Cyanosis. Sweating. Wasting and septic appearance.	More or less characteristic of one or the other form of pneumonia. (See Chapter on Pneumonia.)
Fever .....	Irregular. May not be very high.	Remittent type in broncho-pneumonia. Sustained temperature with sudden onset and crisis in croupous pneumonia.
Respirations .....	Labored, with play of the alæ nasi. Rapid.	Panting or jerky in croupous pneumonia. Labored in broncho-pneumonia, with play of the alæ nasi. Retraction of the xiphoid region may be noted. Rapid in both types.
Circulation.....	Pulse is disproportionate to the fever. Cyanosis at times. There may be dilation of the cervical veins.	Pulse proportionate to the fever. Cyanosis is common in certain forms of broncho-pneumonia.
Nutrition.....	Marked wasting in a relatively short period of time.	Wasting is not so rapid except in cases of chronic or caseous broncho-pneumonia.
Adenopathy.....	May be observed in advanced cases.	Not present, except in cases of tuberculous broncho-pneumonia.
Inspection of the chest.	Marked restriction of movement on the affected side. (Detected by watching the epigastric angle or the play of the inner border of the scapula in respiration.) The study of the "Litten's shadow" may prove more valuable than some authors admit. Filling out or actual bulging of the inter-spaces in advanced cases. Visible apex beat displaced to the left in right-sided empyemata, and pulsation to the right of the sternum in left-sided effusions of size.	Lessened respiratory play of the affected side in croupous pneumonia. In one of the author's cases, there was actual pleurothotonos. Respiratory movement "up and down" and abdominal respiration preponderating in broncho-pneumonia. "Filling out" of the intercostal spaces is never observed. The visible apex beat is not displaced.

	EMPYEMA	PNEUMONIA
Palpation.....	Confirms inspection so far as the limitation of motion and the position of the apex beat are concerned. Usually, there is diminished tactile fremitus below the level of the exudate on the affected side. The sense of resistance over the fluid may also prove valuable. Over the normal lung, one may recognize a wider inspiratory excursion and increased tactile fremitus. Sometimes fremitus is increased even over a large effusion. Local tenderness may be elicited.	In croupous pneumonia there are increased tactile and voice fremitus over the consolidated lobe. With the approach of resolution, there may be marked "rhonchial fremitus." In broncho-pneumonia, rhonchial, tactile and voice fremitus are all increased.
Percussion.....	Light percussion should be employed. The resistance sense is more important than the note. ("Wooden resistance" as opposed to the "rock-like" resistance over a solid growth.) The percussion note is high-pitched, flat and of little volume. Skodaic resonance may be obtained above the level of the effusion. The displaced heart or liver may be detected by percussion alone or by auscultatory percussion. Pitres' coin test may also prove of service. (Tapping with one coin upon another over the affected side, and listening over the other side. <i>Signe du sou.</i> ) Local tenderness may be observed. The Grocco or Rauchfuss sign may be detected on the well side in the presence of a large effusion.	Resistance is increased, though not to the extent present in a pleural effusion. The percussion note is dull rather than flat over consolidated areas. Tympany is valuable as an early sign, and is also present in regions contiguous to consolidated areas in broncho-pneumonia.  No displacement of the heart nor liver. Tympany is a very valuable sign in the diagnosis of pneumonia.
Auscultation.....	Classically, one notes absent or distant breath sounds over the effusion. Often, however, tubular breathing is heard, even over a large effusion. This find must not deceive. Râles, if heard, are usually dependent upon a coexisting lung condition. Egophony.	Early, the breath sounds may be weak. They soon become bronchial in croupous pneumonia and bronchial or broncho-vesicular in broncho-pneumonia. In croupous pneumonia, râles are heard early (crepitant) and late (reduced x-crepitus). In broncho-pneumonia, they are present throughout the disease. Bronchophony.
Blood.....	Leukocytosis, with the polymorphonuclear cells in excess, except in tuberculous empyema. In the latter condition, there may be a lymphocytosis.	Leukocytosis. Lymphocytosis may exist in tuberculous broncho-pneumonia.
Skiagraphy.....	Reveals the effusion. In one of our interlobar cases, it revealed a collection far back, when the physical signs were found anteriorly.	May reveal the consolidated lobe.
Paracentesis.....	Reveals pus.....	No pus.

3. The sooner an empyema is recognized, and the sooner it is treated as a surgical condition, the better is the prognosis.

4. The proper form of treatment has just been indicated. It is surgical, though the surgical measures to be employed may differ in different cases.

5. The most important complicating state is pneumonia. Even when a pneumococcic empyema is recognized early and is promptly treated, the pneumonia may cause a fatal issue. Such a termination was observed in a patient seen with Dr. Eleanor Jones. Some other tuberculous process may likewise cause the death of a patient with a tuberculous empyema. Empyema as a part of a general sepsis has received appropriate mention. Serous pleuritis offers a good outlook, even though it be tuberculous in nature. Aspiration usually betters the patient's chance of recovery.

**Treatment.**—Theoretically, the open-air treatment of pneumonia, improving as it does the resisting powers of the patients, should lessen the frequency of empyemata. It is our impression that patients so treated have fewer complications of any kind. Once recognized as present, the pus of an empyema should be accorded a free exit. In the presence of a small focal collection of pus (interlobar empyema) or of a recent pneumococcic case with mild symptoms of toxemia, a modification of the Heber drainage may be tried. A good-sized trocar and canula is thrust into the dull area; the trocar is withdrawn, a piece of drainage tube is slipped over the canula, and the other end of the tubing is placed under some sterile solution in a bottle. During the past winter, five patients with empyema were treated at the same time on the porch of the Samaritan Hospital. Three of them made good recoveries under this simple method of drainage. If the pus contains streptococci, staphylococci or large masses of fibrin, such drainage will prove wholly inadequate. Thoracotomy, with or without resection of a rib, then becomes necessary. We have been much impressed by the method suggested by Dr. T. Turner Thomas for such cases. We feel that he has answered the problem of successful drainage of the pleural cavity, for he drains from its most dependent portion. In certain old cases of empyema and in some tuberculous cases, far more extensive surgical procedures (such as Schede's operation) may be necessary for cure.

When the pus contains streptococci, we would emphasize the value of antistreptococcic serum in large dosage. Again, with the existence of fistulous tracts in old empyemata, autogenous vaccines have proved

of inestimable service. If such an empyema is tuberculous, tuberculin may hasten recovery.

The after-treatment of empyema is of great importance. It should comprise open-air existence, good nutritious food, and respiratory gymnastics. The well-known James' bottles will prove of much service in hastening the expansion of the crippled lung.

Sero-fibrinous pleuritis may be successfully dealt with in children. As soon as the pleurisy is recognized, we strap the affected side with adhesive plaster, just as surgeons do for a broken rib. The child is promptly relieved of pain, the effusion is less, and the course of the disease is shortened. What if the tendency to adhesion is greater? We doubt that it is; but even granting this, we are taught that repair takes place in this way. Salicylates may prove of value, particularly in rheumatic cases. Rarely do we employ the dry type of diet so often advocated for these patients. If fever is present it may add to their sufferings. Morphine is rarely needed when adhesive straps are employed to limit motion. If effusion occurs, despite the restrictive action of the plaster, paracentesis should be performed. All of the fluid should not be withdrawn at one time. If some is aspirated, the rest is usually absorbed. (See "Blood Pressure Studies.")

## CHAPTER XIV

### DISEASES OF THE GENITO-URINARY SYSTEM

#### THE URINE

Before the time when the child acquires regular habits of urination the most practicable way to procure a specimen of urine for examination is by means of a soft catheter. When this is undesirable or impossible, the child may be placed at short intervals upon the chamber, and hot or cold applications made over the bladder or sacrum to encourage micturition.

The urine may sometimes be caught in small quantities in a cloth or pledget of cotton fastened over the penis or vagina, or, as Holt recommends, a small condom or bottle suspended in such a way that the penis may be introduced into it. A test-tube fitted over the penis also furnishes a good method of obtaining a small specimen. Chapin, in this country, and Hecker abroad have devised special apparatus for this purpose. It is unfortunate that we have no efficient means of collecting a twenty-four hour urine. (Edsall).

**The Quantity.**—The quantity, as in adults, varies between wide limits, depending upon the amount and quality of the food ingested and upon whether a small or a large quantity of water is carried off by way of the skin or the bowels.

Average quantity of urine per diem in  
childhood.<sup>1</sup>

First 24 hours.....	0 to	60 grams ( 0 to 2 ounces)
Second 24 hours.....	10 to	90 grams ( 1/3 to 3 ounces)
3 to 6 days.....	90 to	250 grams ( 3 to 8 ounces)
7 days to 2 months.....	150 to	400 grams ( 5 to 13 ounces)
2 to 6 months.....	210 to	500 grams ( 7 to 16 ounces)
6 months to 2 years.....	250 to	600 grams ( 8 to 20 ounces)
2 to 5 years.....	500 to	800 grams (16 to 26 ounces)
5 to 8 years.....	600 to	1200 grams (20 to 40 ounces)
8 to 14 years.....	1000 to	1500 grams (32 to 48 ounces)

Up to about the third month a healthy infant while awake passes its urine at frequent intervals, often every half-hour or so, but when lying quietly and asleep may retain it for a much longer time—even

<sup>1</sup> This and the following table have been compiled by Emmet Holt from statistics given by Schatanowa, Cruse, Camerer, Pollak, Martin-Ruge, Berti, Schiff, and Herter.

for five or six hours. During the second year the child retains its urine an hour or so longer, and so, as its age advances, the frequency of urination gradually diminishes to the average of adult life.

During the first few days after birth the urine is highly colored, strongly acid in reaction, precipitating in comparatively large quantity uric acid and urates (brick dust deposit is often found upon the diaper, and as we know, "uric acid infarcts" may be present in the kidney); later it becomes paler, of lower specific gravity, but still may be cloudy from the presence of urates and mucus. Hyaline and granular casts may also be found in the new-born (Martin-Ruge). Virchow was the first to show the presence of albumin also at this period.

#### AVERAGE SPECIFIC GRAVITY OF URINE IN CHILDHOOD

1 to 3 days.....	1.010 to 1.012
4 to 10 days.....	1.004 to 1.008
10 days to 6 months.....	1.004 to 1.010
6 months to 2 years.....	1.006 to 1.012
2 to 8 years.....	1.008 to 1.016
8 to 14 years.....	1.012 to 1.020

The phosphates, chlorids, and sulphates are reduced in proportion, gradually increasing in amount with the age.

#### AVERAGE EXCRETION OF UREA PER DIEM IN CHILDHOOD

First day.....	0.076 to 0.114 grams
2 to 7 days.....	0.14 to 0.66 grams
1 to 2 months.....	0.9 to 1.4 grams
3 to 5 years.....	13.09 to 14.01 grams
5 to 13 years.....	16.05 to 21.03 grams

#### RATIO OF UREA TO URIC ACID—(Herter)

New-born <sup>1</sup> .....	14 to 1
First year.....	60-80 to 1
2 to 5 years.....	50-70 to 1
5 to 15 years.....	45-60 to 1

## ANURIA

**Definition.**—Anuria is a complete cessation of the secretion of urine by the kidneys.

**Etiology.**—In rare instances it may be the result of congenital malformation of some portion of the urinary tract. It may occur in the course of fevers or acute nephritis, but here there is more often only an oliguria. Some of the commoner causes are hysteria, shock from fright, traumatism, or operations, after the passage of a catheter or the administration of ether or chloroform, certain poisons, such

<sup>1</sup> From Martin-Ruge.

as arsenic, turpentine, lead, and phosphorus. In collapse during typhoid fever there may be no urine secreted for some time. It may be due to the presence of a calculus blocking one or both ureters.

**Prognosis.**—It will often be seen in infants, when, after a period as long even as twenty hours, the urine will again make its appearance and with the development of no serious symptoms.

As to the length of time a patient may live with absolute suppression of the urine, Herter collected a series of cases in which recovery occurred after periods ranging from four to fourteen days, and Bailey reports an instance of a young girl where, so far as the author could tell, urine was passed but once from October 10th to March 1st. Some doubt, however, is expressed as to the truth of the patient's statements.

**Treatment.**—When the cause is removable, it will receive the first consideration, and the treatment of the condition should be the application of cups or hot fomentations over the kidneys, free administration of purgatives and diaphoretics, followed by diuretics. Large hot irrigations with normal salt solution should be tried, as they stimulate the activity of the kidney in a remarkable way (Osler). This is a particularly useful measure in the anuria of scarlatinal nephritis.

## POLYURIA

**Definition.**—Polyuria is a temporary increase in the secretion of urine. It is merely a symptom and may appear in many conditions.

**Etiology.**—It is produced by drinking large quantities of water, by hysteria or fright, exposure to cold, and by diuretics; it appears in certain forms of Bright's disease and brain lesions, at times in convulsions and acute febrile diseases, and with the resorption of large serous effusions. It is symptomatic of both diabetes mellitus and insipidus.

**Diagnosis.**—It is to be distinguished from diabetes insipidus in that the latter shows a tendency to become chronic in type, and is apt to be associated with changes that are organic rather than functional.

## PHYSIOLOGIC GLYCOSURIA

Glucose is now generally considered to be a constituent of normal urine, but in quantity too small to show a reaction with the older tests, such as Fehling's, etc., but sometimes responding to the phenylhydrazin test. This is of no clinical significance, and therefore phenylhydrazin is inadvisable for ordinary use. It occurs sometimes, however, in the

urine of otherwise healthy children, probably from some faulty metabolism. Sugar, in the form of lactose, is often found in infancy and childhood, and is derived from the milk.

### INDICANURIA

Indol is a product of the putrefaction of albumin from the action of bacteria. It is absorbed and oxidized to indoxyl within the system, where it unites with the sulphuric acid, forming indoxyl-sulphuric acid. The salt of indoxyl-sulphuric acid with which we have to do is the indoxyl-sulphate of potassium (indican). Another substance, indirubin, allied to indican and giving a red color with the same test, is also found, but its clinical significance is unknown.

Indican in very small quantity may be found normally in the urine, seeming to depend upon the character of the food ingested, being increased by an animal diet; but when much exists, it is always pathologic.

**Etiology.**—Indican may be found whenever there is any large accumulation of pus in the body, and when this has been excluded, its presence may be taken to indicate an excessive putrefaction of proteids in the intestine; by far its most frequent source. As an instance of the former may be mentioned gangrenous processes, empyema, and peritonitis, in which it is often a grave prognostic sign. According to Daland, absence of indicanuria almost precludes a diagnosis of peritonitis.

Hochsinger failed to find it in the new-born, and in healthy infants only in traces. He found it in large quantity in most intestinal disturbances, and "always when they were accompanied with acute diarrhea; also in tuberculosis, whether involving the intestinal tract or not." He, as well as Gehlig, ascribed it to the putrefaction of milk proteids in the intestines.

Singer speaks of its presence in urticaria and other skin diseases. Epilepsy, particularly at the time of seizure, and masturbation are said by Herter to be frequently accompanied with indicanuria. The most common conditions, however, with which it is associated are chronic intestinal indigestion and constipation.

**Test.**—Stokvis' modification of Jaffé's test, performed in the following manner, will serve not only as a qualitative, but as a fairly accurate quantitative, test for clinical purposes:

Urine and hydrochloric acid in equal parts are put into a test-tube, to which is added a small quantity of chloroform, *care being*

*taken always to use the same relative quantity of urine, hydrochloric acid, and chloroform; for in this way only can the shades of color of the chloroform as it may vary in different examinations be taken as a guide to the increase or decrease of indican.* To this is added some oxidizing agent, such as Labarraque's solution or solution of sodium hypochlorite, to liberate the indigo; this is then taken up by the chloroform, which assumes a blue color varying in intensity according to the amount of indican present in the urine.

The oxidizing agent should be added one drop at a time, and the test-tube thoroughly shaken, then allowed to stand a few moments for the reaction to take place before adding another drop, for if used too rapidly, the blue color will be bleached when there is only a small quantity of indican present. Slowly in this way all the indigo should be thrown down.

The end of the action may be recognized by the blue color just beginning to fade. If only a trace of indican be present, the chloroform may remain white after the first drop of hypochlorite, when another test should be made, using only the chloroform, and if this fails to show any trace of color, the tube may be set aside for some hours, when it will often be found to show distinctly.

*Precaution.*—Care must be taken to remove any albumin present before applying the test, as it sometimes gives a blue color with hydrochloric acid (Halliburton). Bile also gives a muddy, greenish-blue color to the indican reaction. In decomposed urine the indican may be destroyed. The urine of patients taking bromids or iodids will show with these reagents a somewhat similar reaction, the difference being that in the case of the iodids the chloroform is colored a yellowish red, and in the bromids a reddish-violet tint.

**Treatment.**—The indications for treatment are to control the putrefactive process going on in the intestinal tract.

## ACETONURIA

Acetone has been shown by von Jaksch to be derived from the proteids both of the body and of the food. It occurs in normal urine only in traces, but pathologically may be found in large quantity in many diseased conditions. Most commonly it is seen accompanying the fevers and derangements of digestion, but may be present during starvation, with psychoses (Wagner), as an autointoxication, in advanced stages of diabetes mellitus, and, according to von Jaksch and others, it is of special significance in cases of grave cerebral irrita-

tion. Probably of still greater diagnostic import is its presence in pernicious vomiting. Here it is not only existent in the urine but the odor of acetone is perceived on the breath.

**Diacetic Acid.**—Diacetic acid never occurs in normal urine, but, contrary to the case in adults, it often makes its appearance in fevers, when it is of no serious import. When, however, it is present in diabetes, it is generally a warning of the approach of coma. It is by some held that acetone gives rise to no symptoms, but that they are due to diacetic and oxybutyric acids. Thus, Edsall has viewed pernicious vomiting as an acid intoxication.

## PYURIA

Pus may come from any part of the urinary tract, or, in rare cases, from a perinephritic or perityphlitic abscess opening into some portion of it, but in children its most frequent source is the bladder or the pelvis of the kidney. If from the pelvis, the pus is apt to be in large quantity and perhaps intermittent, but if due to calculus or to tuberculous pyelitis it is continuous. The urine is acid in pyelitis, whereas in cystitis it is usually alkaline and in smaller quantity. Moreover, there will be the characteristic symptoms of cystitis, and washing out the bladder will remove any doubt that may still exist. If from the urethra or vagina, the local symptoms will plainly show its origin.

When the pus comes from a source outside of the urinary tract and opening into it, there will generally be local or constitutional symptoms of its formation, followed by its *sudden* appearance in the urine, and rapidly disappearing within a few days.

The **treatment** is that of the cause.

## HEMATURIA

**Definition.**—Hematuria is the presence in the urine of blood.

**Etiology.**—It may result from the use of drugs, as turpentine, cantharides, and carbolic acid; in hemorrhagic disease of the new-born, hemophilia, purpura, scurvy, scarlatina, typhoid fever, variola, leukemia, malaria, influenza, filaria sanguinis hominis, acute inflammation and congestion, tuberculosis, abscess, and tumors of the *kidneys*, and in renal infarction and calculus; also from any affection or injury of the urinary tract, such as the passage of stone along the *ureters*, the presence in the *bladder* of tumor, stone, or ulceration, and from the

*urethra* in gonorrhea, passage of stone, or use of the catheter. Sometimes renal hemorrhage remains unexplained. The term "renal epistaxis" has been used to describe such cases.

**Urine.**—The color of the urine may vary from normal to dark red, depending, of course, upon the amount of blood present. The "smoky" appearance, when present, is characteristic. The corpuscles may be intimately mixed with the urine, or when this is not the case, may, after the urine has been allowed to stand for some time, settle to the bottom of the vessel, forming a distinct layer.

Under the microscope the corpuscles may appear normal in color and shape, or their form may be totally changed and the color gone, appearing only as faint yellowish rings ("shadow-corpuscles").

These variations help to distinguish the different causes which lead to their appearance in the urine and the portion of the urinary tract in which the hemorrhage has occurred. They are only significant, however, when taken in conjunction with the clinical symptoms and a further examination of the urine.

**Location of Hemorrhage.**—When the blood is in comparatively large quantity, forming, with the urine, a uniform smoky or reddish-brown tint, the corpuscles not separating out and settling to the bottom of the vessel in a more or less well-defined stratum, the hemorrhage may be considered to have taken place in the ureters, pelvis of the kidney, or in the kidney itself; while, on the other hand, when it is from the bladder or urethra, the blood is brighter in color and not thoroughly mixed with the urine.

**Kidneys.**—The urine is generally acid in reaction, of lower specific gravity, and of homogeneous, hazy or dark, color, previously referred to, with perhaps long narrow molds of the ureters. The microscope will show renal epithelium and casts, particularly blood-casts; the blood-corpuscles are variously altered.

Von Jaksch says that when the blood-cells are found to be intimately mixed with the urine and not forming a sediment after standing for some hours, but deeply coloring the urine, and the corpuscles under the microscope appear profoundly altered and the coloring-matter is lost, they probably come from the kidneys themselves, and the symptoms point to acute nephritis or an exacerbation of chronic nephritis. When they appear as few attenuated and washed-out rings, they may have originated in congestive states or in miliary tuberculosis of the kidneys, in conjunction, of course, with other symptoms.

To distinguish between hemorrhage from the *pelvis* or *ureters* is sometimes very difficult. Here the urine is generally acid, and

characteristic epithelium must be looked for under the microscope, together with the physical signs and symptoms. There may also be clots, molded in the shape of the ureters or pelvis.

*The Bladder.*—The blood is brighter in color, clots are more frequent, and the urine is alkaline. The most common cause is calculus, and the blood will be found little, if any, changed from normal, and often coming only at the end of micturition. When ulceration in cystitis gives rise to the hemorrhage, there will be the characteristic symptoms of cystitis: ammoniacal urine, muco-pus, etc.

If uncertainty still exists, the bladder may be washed out, and when, after repeated washings, the fluid comes clear and free from blood, it may be assumed that the source is higher up. The cystoscope may be used, and also catheterization of the ureters resorted to.

In any case, however, the other physical signs and clinical symptoms must be considered before making a positive diagnosis.

**Diagnosis.**—Heller's and Almén's tests, together with the appearance of red blood-corpuscles under the microscope, should prevent any mistake in the diagnosis of hematuria.

**Treatment.**—The treatment is directed to the cause, and is the same as in the adult.

Sometimes in children, when the hematuria is of renal origin, recovery takes place without treatment of any kind except rest.

## HEMOGLOBINURIA

**Definition.**—Hemoglobinuria is the presence in the urine of blood pigment.

**Etiology.**—The destruction of the red blood-corpuscles, setting free, in the blood, their coloring-matter, and thence its passage into the urine, is caused by poisons, such as potassium chlorate, carbolic acid, CO, etc., and by certain diseases: malaria, scarlatina, scurvy, purpura, typhoid fever, yellow fever, and syphilis. There is also an epidemic hemoglobinuria of the new-born (Winckel's disease) and a paroxysmal hemoglobinuria—a periodic appearance in the urine of blood pigment without traceable cause. Sometimes this symptom is displayed by the subjects of inherited syphilis. It may sometimes result from burns, exposure to severe cold, and violent exercise.

**Diagnosis.**—The urine is smoky or even reddish-brown or black in color, acid in reaction, and albuminous. The microscope will reveal masses of pigment and perhaps a few corpuscles, pale in color, but more often the latter will be absent. The urates also may be

stained dark. The spectroscope will give the characteristic absorption bands of methemoglobin or oxyhemoglobin, or both.

**Treatment.**—Treatment is unsatisfactory, but should be directed to the cause, and rest enforced. At times amyl nitrite may be of service.

## ENURESIS

### **Synonym.**—INCONTINENCE OF URINE

**Definition.**—Enuresis is a condition in which the urine is involuntarily discharged from the bladder. It may appear either during the day or night, or both. It may be periodic or continuous. During early infancy—that is, before the termination of the second year of life—enuresis may be described as a physiologic condition, the young infant possessing no control over the bladder. After this time, for a varying period dependent upon the means employed to teach the child to control its bladder, the urine will be evacuated at stated intervals, and the involuntary emptying of the bladder may be regarded as a symptom of some pathologic condition. If infants are trained to use the chamber, the involuntary passage of urine during the day disappears at a very early age. Nightly enuresis, however, never ceases until the second half of the second year, or later.

**Etiology.**—These may be described as organic and functional. Among the former we have malformations of the kidneys, ureters, or bladder, or inflammations of any of these organs. Involuntary evacuation of urine may also be a symptom of various lesions of the brain or spinal cord. In the largest number of cases enuresis is a symptom of innutrition or malnutrition or some disturbance of the equilibrium of metabolism, and symptoms of such disturbances are present in a large number of cases. Instances of this may be found in rachitis or scrofulosis, or, secondarily, may be the results of these conditions or of renal or cardiac disease. In another group the evidences are less pronounced, and in this class we may have intestinal disease, lithuria, polyuria, or diabetes. In a third class the exciting cause may be more obscure, or may manifest itself simply in a disturbance of the nervous equilibrium, shown as a mild or a severe neurosis. We agree with Ludwig Hirt that functional enuresis is a reflex neurosis. In this class of cases many facts gleaned from clinical observation or physiologic research lead us to look for the causes of such disturbances in the highly sensitive nerve-cells. Enuresis has been described as occurring in chorea and some have actually spoken of chorea of the bladder. Some cases are unquestionably of a hysterical

nature (but these are rare). Enuresis may be a manifestation of nocturnal epilepsy.

The physiology of micturition may be described as follows: When the bladder becomes full, an impulse passes up to the cerebral center (see Fig. 58); an inhibitory impulse is then despatched to the sphincter center in the fourth lumbar segment. The impulse then passes out to the sphincter urethræ, that muscle is relaxed, and the patient voluntarily contracts the abdominal walls, squeezing a few drops into the urethra. These drops generate afferent impulses which

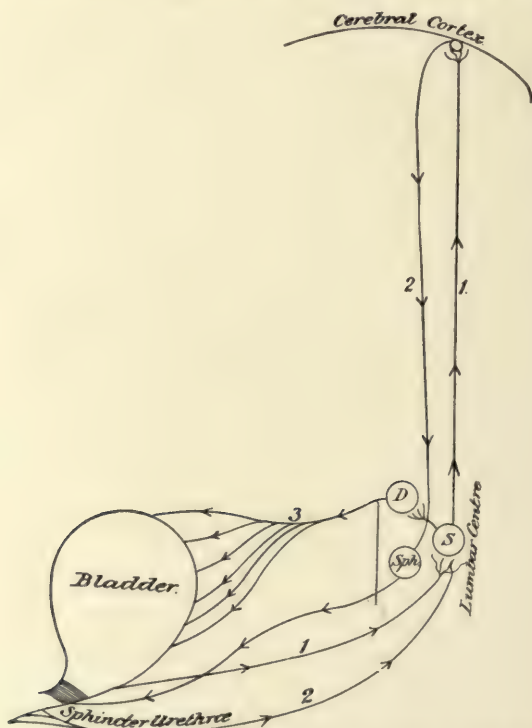


FIG. 58.—DIAGRAM SHOWING REFLEX NERVE ARC OF THE ACT OF MICTURITION.<sup>1</sup>

pass to another center in the lumbar cord—viz., the motor center for the bladder-walls, or the “detrusor center.” Afferent impulses continue, and micturition goes on as a reflex act. It is evident that if we cut off the cerebral arc, we would have the condition seen in enuresis. This, indeed, is the condition found in the idiot or imbecile, or in children in whom a lesion of the sensory or motor portions of the cord exists. In the majority of cases it is probable that the sphincter

<sup>1</sup> For a thorough description of the physiology of micturition see article by Dr. James H. McKee, “University Magazine,” December, 1897.

center in the fourth lumbar segment is at fault (McKee). When the act of evacuation of the bladder occurs during sleep, at which time the inhibitory influence of the wall is in abeyance, the physiology of enuresis is, to some extent, the same as when the cerebral part of the nervous system is at fault through other causes. In some cases the patient may dream of the act, and the evacuation may occur during this dream. At other times the bladder may be emptied without any knowledge of the patient. In the majority of cases the sleep is abnormally long and deep, and here it is probable that the sphincter centers must be at fault. It sometimes happens that the incontinence may be produced by a reflex irritation set up by a local cause, as a vesical calculus, a cystitis, or vulvitis, or in boys, phimosis and balanitis, an elongated or adherent foreskin, and also rectal irritation. Hyperacidity of the urine may be a cause. In one case coming under the care of one of the authors the enuresis could be traced directly to the habitual drinking of coffee.

The **symptoms** consist in the involuntary evacuation of the bladder. If this occurs at night alone we use the term nocturnal enuresis; if during the day diurnal enuresis. The whole bladder may be emptied or simply small amounts may pass "in jets."

The **prognosis** depends entirely upon the cause. In a certain proportion of cases the condition will not last for a very long time, yet in many others success will not be reached except by the long-continued administration of remedies of many sorts, and a careful and thorough investigation as to the cause of the trouble. A certain proportion of cases seem to baffle all our efforts, continuing for months, sometimes with long periods of intermission, during which we are tempted to believe them cured, only to find the incontinence returning. Some continue to the age of puberty when the symptom suddenly disappears. A case of enuresis should not be considered cured until several months, or even a year, elapse without the return of the condition.

**Treatment.**—No matter what form of treatment is used, the results are often discouraging. Patients will temporarily improve; indeed, may seem to be cured, only to lapse again into the old condition. On the other hand, spontaneous cures are sometimes seen. Occasionally they may seem to result from nothing more than a change of surroundings or of diet. Buckingham speaks of a boy upon whom various methods of treatment had been tried who was cured during a vacation in which he cultivated a taste for athletics. There is no class of cases in which the etiology should be more carefully studied

than those affected by enuresis. Central and spinal lesions and various malformations of the urinary apparatus must be sought for. The urine must be frequently examined, and if found to be highly concentrated, the patient may be greatly benefited—in some cases absolutely cured—by simply diluting it by increasing the amount of fluids. When phimosis is a cause, it should be treated by the methods before given. Some instances of nocturnal incontinence of urine may be prevented by making the child empty the bladder thoroughly just before retiring for the night; or if the incontinence occurs during deep sleep, the child may be awakened once or twice during the night to empty the bladder. There are instances, too, where the sleep is light and shallow, and these are relieved by sulphonal at night. When the patient has a highly sensitive vesical sphincter, it may be necessary to raise the foot of the bed so as to keep the urine from pressing against that part. A regular mode of life, with outdoor exercise, is especially beneficial. In cases of lowered nerve action strychnin is indicated, while in the opposite class of cases, those with extreme irritability of the nerve centers, bromids, atropin, and belladonna are the indicated remedies. These drugs should be given until some decided symptoms of their action are manifested. In diseases attended by congestion the use of ergot has been recommended. Cold douches to the perineum and faradism have been of some use. The passage of a cold sound in boys is recommended. Urethral electrization is often efficacious. Increasing doses of atropin or hyoscin hydrobromate are the most reliable measures for the relief of the symptom.

### VESICAL CALCULUS

In children three varieties of stone are met. The form of calculus in the bladder most frequently found is that known as the uric acid formation, which, according to statistics quoted by J. William White, composes from two-thirds to five-sixths of all calculi found in the kidneys, ureters or bladders of children. This variety may be made up entirely of uric acid, or may be composite, containing also oxalate of lime and the urates of ammonium and sodium. The uric acid stone is usually not very large and of oval shape, varying in color from a yellowish-white to a dark brown. Externally this form of calculus may be smooth or slightly roughened, while on section it is found to be laminated or amorphous. The laminated variety is extremely hard, and capable of a high polish. The amorphous form is easily broken, and resembles coarse sand.

The second variety is composed of oxalate of lime, and is sometimes called the mulberry calculus. It is generally round, covered with small spicules, and its color varies from gray to brownish-black.

The third variety, known as the urate of ammonium calculus, is only occasionally found. It is a smooth, flat, oval stone, brittle, and of a yellowish color. Other varieties of calculus, such as the mixed phosphate or the ammoniomagnesium phosphate and those composed principally of phosphate or carbonate of lime, are occasionally seen. Cystic oxid, xanthic oxid, and other varieties of stone have been described, but are extremely rare. The size of vesical calculi varies widely. They may be single or multiple, and are described as being *free* when found loose within the bladder, and *attached* when held to the bladder-wall either by a fold of mucous membrane or a band of lymph (White).

**Etiology.**—But little is known as to the cause of cystic calculus in children. It is possible that an inherited gouty tendency may have something to do with causation, but, on the other hand, it has been demonstrated many times that children of the poor, or those having few of the luxuries or even the necessities of life, are more frequently affected than children born in better circumstances. Boys are more subject to stone than girls. A predisposition seems to run in certain families, and the inhabitants of some locations are more frequently affected than those of others. It might be supposed that the water-supply of a certain location could have something to do with this, yet it has been proved many times that it has not. Race seems to have some influence, it having been shown that the negro is affected far less frequently than the white races.

In almost every case stone has its origin in the kidney, and it is believed that the uric acid infarcts so often seen in the kidneys of newly born children form the first stage in the production of calculus, and that the large quantity of uric acid which is present in early life will explain the frequency of this form of calculus, or its presence may act as a nucleus of other forms of calculi which may later develop in the bladder. Occasionally the stone may be formed around a nucleus which is composed of some foreign body which has been passed into the bladder. This is, however, very rare.

**Symptoms.**—The group of symptoms which points to the presence of renal calculus is in most respects the same in children as in adults. In many cases the first evidence of its presence will be shown by an attack of intense pain, accompanying a group of symptoms known as nephritic colic. In the midst of health the child is suddenly

attacked by violent pain, first felt in the lumbar or hypochondriac region, and extending toward the scrotum and end of the penis. The testicle on the side affected will be drawn up by a spasm of the cremaster muscle. Pain is also felt in the groin and thigh on the affected side. The patient has a continual desire to pass water. The attack of colic is accompanied by sweating, which is profuse, and the skin becomes cold and clammy. Pain may be so intense as to cause convulsions and collapse. The urine is passed frequently, but in small amounts, and is very highly colored. Actual suppression may occur as the stone makes its progress along the ureter, and will only cease permanently when it has reached the bladder, at which time the pain also ceases suddenly. After the stone has reached the bladder, the foregoing group of symptoms gives place to others.

The principal symptom of stone in the bladder is increased frequency of urination. The desire to evacuate the bladder may be continuous, amounting to actual incontinence. This symptom is worse by day, when the patient is up and around, than at night. It is increased by motion, and its amount depends to a certain extent on the shape of the stone. Occasionally retention of urine occurs. This may be produced either by the stone acting as a ball-valve and stopping the flow of urine, or, in some rare instances, is reflex. The pain is of a darting, burning character, increased during urination, particularly at the termination. It is caused by the mucous membrane of the bladder coming in contact with the stone. The pain, however, is rarely felt in the bladder, but is referred to the under surface of the penis some distance behind the external meatus. It is sometimes so severe as to cause convulsions. It continues until a sufficient amount of urine has collected in the bladder to raise the mucous membrane away from the stone. Various reflex pains, referred to different parts of the body, are not infrequently associated with vesical calculus; these may be felt in the rectum or perineum, and occasionally in various parts of the body far removed from the seat of the trouble. From the constant irritation of the penis the habit of masturbation may be set up, or from handling of the parts the foreskin may be rendered abnormally long, while in some cases phimosis or paraphimosis is produced by this cause. Cystitis is almost always present, with its characteristic urinary finds. From the constant efforts at straining prolapse of the rectum may be produced. When the flow of urine is stopped by the calculus obstructing the vesical orifice, the child may assume various peculiar positions while evacuating the bladder, in order to cause the stone to fall away from the opening of

the urethra and allow the passage of urine. Hematuria may occur, but is rarer in children than in adults. In our experience, it is a more common symptom in renal, or ureteral calculus than in vesical stone. As sequelæ we very frequently note chronic cystitis, and sometimes pyelitis. Dilatation of the ureters may occur, and from extension of inflammation there may arise nephritis or even suppurative pyelitis or pyelonephritis.

**Diagnosis.**—Stone in the bladder may be confounded with one of several conditions. The diseases most likely to be mistaken for it are a contraction of the urinary meatus, cystitis (from other cause) phimosis, the condition known as irritable bladder, vesical tumors, and renal calculus. None of these conditions will, however, show a group of symptoms so severe and well marked as stone. The surest factors in diagnosis, however, are the finding of the stone by examination with a sound, a cystoscope or the X-ray apparatus.

In all cases of irritability of the bladder in children, especially when there is pain during or immediately following the act of urination, and when phimosis or malformation of the penis and urethra does not exist, the bladder should be carefully examined for stone. If the urethral sound is used it is best to administer an anesthetic, in order to produce complete relaxation of the part and to prevent the child from struggling and possibly receiving injury at the time of examination. The bladder should then be filled with warm solution of boric acid, to distend the walls and give greater space for the movements of the searcher. By the careful use of a sound in the bladder the presence of a stone can readily be demonstrated by the characteristic "click" which the instrument makes in coming in contact with the stone. Sometimes this sound can be heard some distance from the patient, particularly if a sounding board is used on the distal extremity of the sound. In experienced hands, the cystoscope may yield most positive information. In view of several experiences, however, it is our view that an X-ray examination should precede either of these other methods.

The **treatment** is surgical: either by crushing (lithotrity) or lithotomy. If the stone be small, or even of moderate size, it may be crushed by a lithotrite and evacuated at one sitting—as in done in adults. If however, the stone is very hard, it may be better to do a suprapubic cystotomy. The lithotrite has been broken in experienced hands. Of late years this mode of operating has been very extensively practised, with admirable results. Piperazin as a solvent of uric acid stone has been largely used, with conflicting reports as to results. D. D. Stewart has reported toxic symptoms from its employment in adults, and recommends, for a child of ten years, that the dose be not over  $2\frac{1}{2}$  grains.

## CYSTITIS

Inflammation of the mucous membrane of the bladder is much rarer during infancy and childhood than in adult life.

**Etiology.**—The most common cause of vesical inflammation is stone. It may also be produced by a retention of urine within the bladder, caused by a contracted meatus and from phimosis. The long-continued administration of irritating drugs may produce it, or a growth within the meatus may act as a causal factor. A form of very severe cystitis may appear in the course of tuberculosis in children, and is due to tuberculous infection of the bladder. Occasionally it is produced by an extension into the bladder of any infection from without, particularly in girls. The colon bacillus is the most common bacteriologic cause in female infants. Vulvovaginitis may be the cause of urethritis and cystitis as in the female adult. It occasionally appears in the course of infectious diseases, particularly in typhoid fever and in epidemic influenza. We have seen it in both diseases.

**Symptoms.**—The symptoms of cystitis in children are practically the same as those in the adult. There is some rise of temperature and fretfulness. Indeed, the child may appear quite ill. Micturition is frequent and painful, the urine, which is passed in small quantities, is viscid, from pus and mucus, alkaline or mildly acid in reaction, and, if acid when voided, promptly undergoes decomposition, becoming strongly alkaline, converting the contained pus into a thick, mucilaginous mass—a characteristic and diagnostic sign. The microscope will show crystals of triple phosphates and amorphous phosphates of lime, pus-cells, bladder, epithelium, many bacteria, and, in the severer cases, especially caused by calculus, blood-corpuscles. In typhoid fever, the urine may contain many typhoid bacilli and cystitis may result as stated.

*Chronic cystitis* arises very commonly from stone in the bladder or the presence of foreign bodies—a tumor—or tuberculosis. The acute form, unmitigated by treatment, may progress into the chronic variety.

The *symptoms* are painful and frequent micturition, the passage of urine later becoming almost constant, and associated with an irritation of the external genital organs, caused by an ammoniacal condition of the urine. Prolapse of the rectum is very commonly associated, from constant straining, when the irritation is caused by the presence of a stone. The urine is subject to the same changes as in the acute form.

**Prognosis.**—In the acute form the prognosis is good, providing the cause can be removed. When prompt treatment is not instituted, the condition rapidly passes into the chronic form, which is very persistent and hard to control. The outlook for cure in the chronic form depends very much upon the cause and the duration of the attack. It must not be forgotten that cystitis may appear as a secondary consequence of diseases of the kidneys.

**Treatment.**—The cause of the disease must always be sought for and removed if possible. The patient should be kept at rest in bed. The diet should consist of milk. Water should be given freely, and various diluent drinks, such as flaxseed tea, mucilage of acacia, and the citrate or acetate of potash, may also be made use of. The bowels should be thoroughly opened by salines. Poultices or hot fomentations should be placed over the bladder. If there is much pain, opium may be used by suppository or enema, but it must be remembered that this drug should be given with great caution to children. Various other agents may be employed, such as tincture of aconite, the spirits of nitrous ether, benzoate of sodium, etc. Tyson recommends, when great irritation is present, injections into the bladder of cocain: for an adult not more than two grains at one time. This must be used cautiously in children and in much smaller doses. Belladonna often relieves vesical spasm. We are exceedingly fond of the following efficient though somewhat disagreeable combination:

R. Sodii bromidi.....	℥i
Sodii benzoat.....	℥ij
Tr. belladonna.....	fl. ℥i—fl. ℥ij
Glycerin.....	fl. ℥ij
Aq. camphor.....	q. s. ad ft. fl. ℥iij
M. et solve.	

Sig.—fl. ℥i every three hours. (Dose for a six-year-old child.)

In chronic cystitis the bladder should be washed out once or even twice daily, if necessary, with warm water, to which may be added boric acid, one dram to the pint. It should be injected slowly in small quantities at a time, according to the capacity of the child's bladder, and repeated until the water comes away clear. The water should be a temperature of about 100° F. In chronic cystitis nitrate of silver irrigations (solution of 1-2000) may prove of great service. Antogenous bacterins have also produced cure. Urotropin is useful in older children, in doses of one to five grains (according to age), three or four times daily, well diluted. Urotropin should always be administered to typhoid patients to clear their urine of bacilli. Its adminis-

tration must be watched carefully however. Salol is another very efficient urinary antiseptic, being eliminated as salicylic and carbolic acids. The physiological action of salol should be carefully watched in children for while it is an efficient remedy one of us has seen considerable depression follow its use.

### PHYSIOLOGIC ALBUMINURIA

**Synonyms.**—CYCLIC ALBUMINURIA; FUNCTIONAL ALBUMINURIA; ORTHOTIC ALBUMINURIA (HEUBNER)

It is pretty generally held now that occasionally there may be an appearance of albumin in the urine that is physiologic, but before such a diagnosis is made care must be taken, first, that in performing the tests it is serum albumin that is found and not any of the other proteid bodies, such, for instance, as nucleo-albumin. This may seem at first sight a needless caution, but with some of the more recent and sensitive reagents a reaction similar to albumin is sometimes shown with even distilled water which has been passed through Swedish filter-paper, the albumin of the paper being responsible for it.

Furthermore, the albumin in these cases is always in small amount. The examinations must be continued over a considerable period of time, and the urine should show no other changes from the normal, such as casts, and the elimination of urea must show little or no diminution in quantity; and further, to exclude the possibility of any renal changes, there should be found no such characteristic symptoms as dropsy (in any degree), a pulse of high tension, nor evidence of cardiac hypertrophy. By some it is claimed that no alteration has taken place in the epithelium lining the capillaries of the tufts or of the glomeruli, while others contend that it is always in a state of "cloudy swelling."

There occurs what is termed a "*cyclic albuminuria*," where albumin can be demonstrated during the day but is absent from the urine secreted during the night. The erect posture has been offered as an explanation (Pavy, etc.). There is an occasional albuminuria that is associated with an oxaluria, and Trissier has noticed it in young children of gouty parentage, generally boys.

Among the causes said to produce it are an albuminous diet, violent exercise or emotion, and cold bathing. At times there may be no traceable cause.

**Treatment.**—The treatment consists in the removal of the cause, where one exists, and the building up of the patient on general lines.

## DISEASES OF THE KIDNEY

Before taking up a consideration of inflammations and various other diseased conditions of the kidney structure, it is necessary to make a few preliminary observations: We are free to confess that we have not been able to pursue with success, some methods of physical examination as our brethren on the other side of the Atlantic have. Nevertheless, when such men as Israel are able to detect slight tumefactions, etc., by means of palpation, such methods are certainly to be commended to the student.

Inspection may certainly prove of value in the cases of renal growths: hydronephrosis, and para-, peri-, or epinephritis (Strauss). Bulging in the renal region posteriorly and laterally would strongly suggest the first two conditions, while boggiess and redness of the integument in this region should make us think of suppurative conditions in the neighborhood of the kidney.

Palpation is certainly a valuable method of examination in the presence of renal growths. One may not only outline the tumor limits; but may also study the consistency of the sarcomata, cysts, etc. Years ago Dr. Edward Martin, called our attention to the importance of tenderness in the "two kidney triangles" (under the twelfth rib posteriorly and below the costal margin anteriorly) in renal calculus, perinephritic abscess, etc. Langstein (Pfaundler and Schlossman) recommends that one palpate for tenderness, position, size, mobility and tumors in investigating the kidney. He quotes Israel as recommending a position for the patient midway between the dorsal and the full lateral position. One hand is then placed posteriorly over the renal region, and the other anteriorly. Rectal examination, particularly under anesthesia, may enable one to recognize the lower pole of an enlarged kidney and to detect a retroperitoneal growth. (Sarcoma of the kidney or retroperitoneal glands, etc.).

With percussion we have not had uniform success except in cases of tumors as floating kidney, though Steffen believes it quite possible to outline the size and shape of the normal kidney by percussion.

The use of the X-ray may reveal information of a most positive character. Only recently, one of us saw a calculus removed from one ureter, when the symptoms had pointed strongly toward renal tuberculosis. Two Roentgenograms, revealing the shadow in exactly the same location, enabled us to make the proper diagnosis. Tumors also should be studied in this way. In sufficiently expert hands, ureteral catheterization may be employed in older children.

Upon the importance of urinary analyses, we need scarcely lay additional stress, except to say that the need for such is probably greater in early life than in adult years. Chemical examinations alone may fail to reveal important conditions, like the presence of pus, etc., and should be supplemented by microscopic studies.

### ANOMALIES OF THE KIDNEY

Most of these are rare. There have been described, however:

1. Absence of the kidneys or of the whole urinary tract (very rare). Only one may be absent.
2. Unilateral malformations, usually affecting the left kidney. (Hyperplasia of one kidney usually means hypoplasia of the other.)
3. Multiformations of the kidney or ureters (very rare).
4. Misplacements of the kidney (dystopia). One of us has seen a kidney in the true pelvis situated between the psoas and iliacus muscles.
5. Fusion of the kidneys—horse-shoe kidney.
6. Cystic kidneys as hydronephrosis, either one of which may be observed alone or in combination with the other.
7. Movable kidney.

Holt classifies the twenty cases that have come under his notice as follows:

Fusion of the kidney (horse-shoe)	4 cases
Supernumerary ureters	4 cases
Hydronephrosis (alone)	8 cases
Cystic degeneration of the kidney (alone)	2 cases
Hydronephrosis and cystic kidney	1 case
Single kidney	1 case

### NEPHRITIS

A great many difficulties meet one in attempting to classify the inflammations of the kidney structure. Porter has given a very complicated classification in which many varieties are recognized pathologically and clinically. McFarland, on the other hand, gives the old and relatively simple classification. He contends that the organism is supplied with many times the amount of secreting kidney surface that is actually needed. Thus patients may live for a long time, be in relatively good health, and yet show at autopsy much kidney change in certain areas. Again, in one case, one tissue may be affected more markedly, in another some other tissue. Langstein agrees with Ponfick that an etiologic classification would be advisable. He suggests the following, admitting, however, that a complete etiologic classification is not possible at the present day:

1. *Nephritis of Infancy:*

1. Of gastrointestinal origin.
2. Due to other infections and intoxications.
3. Due to congenital syphilis.
4. Contracted kidney.

2. *Nephritis of Older Children:*

1. Scarlatinal nephritis.
2. Diphtheritic nephritis.
3. Due to other infections and intoxications and of unknown origin.

We quite agree with McFarland and others, however, that it is probably best to make a classification as simple as possible and speak only of acute and chronic nephritis. Properly speaking, both pyelitis and pyelonephritis should be included under this heading, though we shall consider them separately.

## ACUTE CONGESTION OF THE KIDNEY

**Synonyms.**—RENAL HYPEREMIA; ACUTE RENAL CATARRH

**Etiology.**—Acute renal congestion may occur as the result of traumatism from the use of certain irritating drugs—such as turpentine or cantharides—or from cold. The most common cause is the acute infectious diseases.

**Pathology.**—The blood-vessels of the kidney are engorged with blood, and, owing to this congestion, there is frequently an escape of serum, red blood-cells, and leukocytes. The epithelium of the parenchyma is in a state of “cloudy swelling.”

**Symptoms.**—The symptoms of renal congestion itself are rather varied. There are generally some headache, pain in the back, and general malaise. The urine is scanty, highly colored, from the presence of red blood-cells or blood-casts, and of high specific gravity. It always contains some albumin. This may differ markedly in amount. The duration of the attack may vary considerably.

The **prognosis** will depend entirely upon the cause of the congestion and whether or not the congestion proves to be the beginning of an attack of acute nephritis.

**Treatment.**—The bowels should be freely opened, preferably by the use of salines or calomel; the latter is especially useful on account of its diuretic action. Hot vapor baths should be employed to produce diaphoresis. Mild counterirritation over the kidneys by means of dry cups or hot poultices is to be employed. Hot enteroclyses

(110°) are also indicated. When irritability of the bladder is present camphor may be used, or in some cases small doses of camphorated tincture of opium may be given with benefit.

### CHRONIC CONGESTION OF THE KIDNEY

**Etiology.**—Chronic renal congestion may be produced by the continuance of the acute form. It is most frequently met as the result of an interference with the return circulation of the kidney, which may appear during the course of heart disease. It is also very common in chronic bronchopneumonia, or chronic pleurisy. It may be found when this circulation is interfered with by reason of an abdominal tumor, also by thrombosis of the renal vein.

**Pathology.**—The kidneys are enlarged in early stage; later they become normal in size, darker colored than normal, and of firmer consistency. The entire organ is distended with blood. The capillary vessels are engorged, their walls being thickened.

**Symptoms.**—General dropsy is a common feature, together with the symptoms of the disease causing it. The urine is always scanty and of high specific gravity, containing usually a small amount of albumin. The microscope will reveal the presence of a very few small hyaline (cylindroids) or granular casts. These may not, however, be regularly present. The bowels are generally constipated; irritability of the bladder is commonly present. Uremia is very infrequent.

**Treatment.**—In addition to the treatment of the condition causing the congestion, an effort should be made to increase the amount of urine by the use of digitalis, especially the infusion; by sweet spirits of niter, the alkaline diuretics, caffeine and other drugs of the same order. The bowels should be kept open by salines and hot baths, or vapor baths should be used to promote diaphoresis. When the amount of urine passed is extremely small, nitroglycerin may be used with benefit. Suitable dietetic and hygienic measures should, of course, be employed.

### ACUTE DEGENERATION OF THE KIDNEY (CLOUDY SWELLING)

#### **Synonyms.**—FEBRILE ALBUMINURIA TOXIC NEPHRITIS

**Etiology.**—Acute degeneration of the kidneys is very frequently present in the course of acute infectious diseases, but it is found oftenest and is most marked in cases of diphtheria, scarlet fever, and acute pleural pneumonia. It may appear during the course of any disease accompanied by long-continued high temperature. In all probability it

is caused by direct irritation of the epithelium of the tubules by toxins eliminated by the kidneys. Under such conditions, it might be more scientific to speak of toxic nephritis. Irritating drugs may also produce it.

**Pathology.**—The color of the kidney is pale, and the whole organ is somewhat enlarged. The cortex is thickened, and the straight tubules are marked by yellowish-gray lines. The epithelium of the tubules undergoes granular degeneration. Some exudation of serum may take place.

**Symptoms.**—There are no special symptoms connected with this form of renal disease other than those which accompany diseases producing it. The urine will contain a moderate amount of albumin and sometimes a few granular and hyaline casts.

**Treatment** should consist in the use of a liquid diet and diuretics, in addition to the treatment of the condition producing the degeneration.

#### ACUTE DIFFUSE NEPHRITIS

**Synonyms.**—ACUTE GLOMERULONEPHRITIS; ACUTE PARENCHYMATOUS NEPHRITIS; ACUTE DESQUAMATIVE NEPHRITIS; ACUTE TUBULAR NEPHRITIS; ACUTE EXUDATIVE NEPHRITIS; ACUTE INTERSTITIAL NEPHRITIS

**Etiology.**—In the majority of cases acute diffuse nephritis follows an attack of one of the infectious diseases, particularly scarlet fever, in which case it is generally admitted that the exciting cause can be attributed to the scarlatinal poison, probably the result of direct irritation from the toxins of the disease. It is highly probable too that bacteria (streptococci, etc.) play important rôles in this as they may in the other infectious diseases. The frequency of the disease as a sequela of scarlet fever varies considerably with epidemics. It sometimes follows diphtheria, when the exciting cause is probably toxic, the action of the poison being similar to the scarlatinal complication. Some of the other diseases in which acute nephritis has been observed are typhoid fever, pneumonia, influenza, smallpox, pertussis, varicella, tonsillitis, measles and various septicemic and pyemic states. Koplik states that the following bacteria have been found in kidney lesions *Diplococcus pneumoniae*; typhoid bacillus; streptococci of various sorts; staphylococci, and the bacillus *pyocyaneus*. Syphilis probably deserves special mention as an etiologic factor. (See Etiologic Classification.) Certain irritating drugs may also produce nephritis (ether, cantharides, urotropin, etc.). Cold and exposure have been attributed as causes,

and in some cases the etiology is obscure. As predisposing causes of the postscarlatinal variety it has been stated that allowing the patient to get up too soon after the disease or the too early administration of solid foods has a tendency to aid in the irritation of kidneys previously weakened by the scarlatinal poison. Blows and injuries to the back have also been given as causes. Acute diffuse nephritis is much more frequently seen in children and young adults than in the old.

**Pathology.**—The main points in the morbid anatomy of the kidneys are as follows: The entire organ is enlarged, often to a considerable extent, and is softer than normal. The capsule strips quite readily. In the early stages of the disease the kidneys are sometimes considerably congested, but after the disease is well established, they are a yellowish-white color mottled with red. Thickening of the cortex occurs, this portion being usually yellow, and showing a distinct contrast to the pyramids, which are red. Microscopic examination shows the characteristic changes of this variety of nephritis, which consist in the formation of connective-tissue cells in the stroma and proliferation of the cells forming the capsule of the Malpighian bodies. The longer the duration of the disease, the denser and more fibrous in character will the connective tissue appear. Finally, the glomeruli undergo permanent change; the tufts will be reduced by the growth of the endothelial cells lining the capsule, which may ultimately form new fibrous tissue. The tubules often contain red blood cells, leukocytes and desquamated endothelium and casts. The cells of the distal and proximal convoluted tubules also exhibit more or less marked degeneration. Round-cell infiltration is noted in the interstitial tissues. Hemorrhages may occur, particularly into the cortical structures.

The widespread nature of these lesions and the fact that in certain cases, the changes preponderate in certain tissues, explain why so many different investigators have described different forms of acute nephritis, applying different terminologies. Thus, if the most marked lesions are observed in the glomeruli, they employ the term glomerular nephritis; if in the tubuli contorti, tubular nephritis; if exudation is particularly marked, exudative nephritis; if hemorrhages occur (influenza, etc.), hemorrhagic nephritis, etc.

Heubner has stated that the blood-vessels (especially of the glomeruli) were principally affected by the scarlatinal poison; the epithelium of the tubules particularly by the toxin of diphtheria, and the interstitial tissues by septic processes. This is only a generalization, however, as the work of Councilman, Mallory and Wright has shown.

**Symptoms.**—As a rule, the urine is considerably diminished in quantity; indeed, suppression is not uncommon. Albumin is always present in large amounts. The color of the urine is smoky or reddish brown, due to the presence of red blood-globules or hemoglobin. The specific gravity is at first high, but later it may be low; the amount of urea eliminated is below normal. The microscope shows the presence of casts in great variety. Hyaline, granular, and epithelial casts are always present, and pus-, mucus-, and blood-casts are not rare.



FIG. 59.—NEPHRITIS OF UNKNOWN ORIGIN. The child was under observation for a year, during which time albumin and casts persisted. She had some edema most of this time and at periods anasarca. Evidentially, however, she made a clinical recovery.—(*Services of Drs. Hamill and McKee, Philadelphia Polyclinic Hospital.*)

Chlorids and earthy phosphates are at first diminished. Hematin, indican, and uric acid show an increase (Tyson). Leukocytes and red blood-cells, with a large variety of cells from the renal epithelium, are present. Headache, backache, fever of a moderate grade, high blood pressure and stupor or extreme restlessness are common symptoms. In cases of ordinary severity which tend toward a favorable termination the symptoms should subside in from one to three weeks. The edema gradually passes away and the temperature returns to the normal. The quantity of urine increases, and the amount of urea excreted, which during the attack has been much



FIG. 60.—NEPHRITIS FOLLOWING PERTUSSIS IN A THREE-YEAR-OLD BOY. He lived nearly eight months after albumin and casts appeared in his urine. The urinary fluids and edema never completely disappeared. Twice when he was profoundly uremic, he was relieved by peritoneal paracentesis. Finally, cortical decapsulation (Edebohl's operation) was performed by Dr. Babcock, under spinal anesthesia. The patient stood the operation well; but succumbed to uremia one week later. Despite the length of time his nephritis had lasted, Dr. McCormell's pathologic study revealed acute parenchymatous nephritis.—(*The Samaritan Hospital*.)

below the average, gradually becomes greater, while the amount of albumin and the number and variety of casts which have been present during the disease begin to decrease. It should not be forgotten that it is quite possible that a few casts and a trace of albumin may persist for a considerable time. The disease, however, may increase in severity, the temperature continue high, the pulse become full, rapid, and of high tension. The urine, which before was scanty, may now be actually suppressed and symptoms of uremia follow. The attack of uremic poisoning may begin with symptoms of restlessness or apathy, headache, nausea, and vomiting, the vomit often having the odor of urine. Dimness of vision is sometimes seen but albuminuric-retinitis, in childhood, is exceedingly rare in any form of nephritis. Amaurosis (probably of central origin) is not so uncommon. Later the patient may pass into a state of stupor or coma or have convulsions. Diarrhea is not an uncommon early symptom in children.

**Complications and Sequelæ.**—The most frequent complications are pneumonia, pleurisy, edema of the lung, pericarditis, and endocarditis. Occasionally, though rarely, meningitis and edema of the glottis may complicate the disease.

**Prognosis.**—In so far as the recovery from the acute attack is concerned the prognosis is guardedly good; the majority of patients recover. There is, however, considerable danger of the disease progressing into the chronic form, in which the outlook for absolute recovery is not so favorable. The existence of severe nervous symptoms, stupor, intense headache, dimness of vision, or the appearance of uremia, would make the outlook less hopeful. The urine should be carefully examined at frequent intervals, as the amount of albumin and the number and variety of the casts are valuable aids in prognosis. Patients suffering from this disease require constant watching for a long period of time. When the case progresses into chronic nephritis, the outlook, although rather doubtful as to the chances for absolute cure, is by no means hopeless so far as the life and comfort of the patient go. With care and attention to diet and to the general rules of life, many of these patients live for many years. Relapses are frequent, and death may occur during one of these, or the patient perish from pneumonia, edema of the lungs, or from some intercurrent malady.

**Treatment.**—Of primary importance in the therapeutics of acute diffuse nephritis is the stimulation of the skin as the most important adjunct in eliminating the excrementitious substances of the body and thus aiding the crippled kidneys. For this purpose frequent sponging

with hot water, warm baths, or, what is often better, repeated hot packs, or hot air baths are of the greatest use. There may also be given sweet spirits of niter, with or without very small doses of ipecacuanha; also the fluid extract of jaborandi, in doses of from five to ten minims, repeated as often as may be necessary at intervals of from two to three hours. Poultices to the lumbar region are often efficacious. The bowels must be kept freely opened by salines and calomel. These should be given in quantities sufficient to produce two or three movements daily. The urine should be diluted as much as possible in order to decrease its irritating properties. With this in view the patient should be made to drink two or three glasses a day of filtered water, or, if it is preferable, with the addition of about twenty grains of bicarbonate of sodium to each glass. With the injections (rectal or subcutaneous) of sodium bicarbonate and sodium chloride in solution, a treatment suggested by Fischer, we have had no experience. Some have published favorable reports on this treatment, while others have criticized it severely. Some authorities recommend that two or three grains of the citrate of potassium be added to a glass of water. The diet should be fluid, preferably milk. Morse suggests, and we quite agree with him, that formulæ feeding, in which the proteids are low, would seem more rational than feeding whole milk, buttermilk, etc. The fats may often be given in relatively high percentages. Should milk not be well borne, then such preparations as whey, buttermilk, koumiss, or junket may be used. If the nephritis follows an attack of scarlet fever, it is generally recommended that the patient be kept in bed for at least a month after the temperature has become normal. In severe cases, where the fever is high, the urine scanty, and the amount of edema considerable, diaphoresis should be maintained by the use of the hot pack or vapor bath. Pilocarpin may be used hypodermically, and be given in doses of  $1/60$  of a grain to a child of three or four years. In order to guard against the depressing effects of the drug, stimulants should be conjointly given. One of the simplest and at the same time a most effective method of producing diaphoresis is to place under the bedclothing, at the feet of the patient, a very hot brick, and upon it pour about two ounces of alcohol, thus producing an alcohol vapor bath. This alone will often bring about a most profuse sweating (E. L. Duer). Counterirritation should be applied over the kidneys by poultices, a mustard plaster or dry cups. In cases where symptoms of uremia occur, the temperature being high, nitroglycerin should be given in quantities sufficient to produce the effects of the drug. Holt recommends that  $1/300$  of a grain be given

every hour for three or four doses. In some cases of uremic convulsions, injections of morphin may be of service.

Venesection has also been recommended as a means of rapid depletion when the symptoms are urgent. For the anemia which is very commonly seen iron is required. When the disease has existed for some time or has passed into the subacute form, the patient had best be sent to a warm, dry climate, especially during the winter months. Flannel underclothing ought to be worn next the skin, and every precaution taken to prevent the patient taking cold.

### CHRONIC NEPHRITIS

Under the title of chronic nephritis will be described three forms of chronic inflammation of the kidney structure, all of which are rarely seen in children, yet occur with sufficient frequency to warrant at least passing notice. The varieties of chronic nephritis are, first, *chronic diffuse nephritis with exudation*, known also as *chronic parenchymatous nephritis* or the *large white kidney of Bright*; second, the so-called *waxy* or *lardaceous kidney*; third, *chronic diffuse nephritis without exudation*, known also as granular kidney, sclerosis of the kidney, contracted kidney, or chronic interstitial nephritis.

**Etiology.**—The most frequent cause of the first variety is a continuation of the nephritis following an attack of scarlet fever or other disease of the same class; in fact, the etiology of this form of chronic renal inflammation is practically the same as that of the preceding form described. Waxy kidney is most apt to follow prolonged suppuration, especially that accompanying disease of the bones and joints. True chronic interstitial nephritis—the variety described under the second heading—is exceedingly rare in childhood. When occurring, the causes are generally hereditary syphilis, tuberculosis, alcoholism, and chronic valvular diseases of the heart. Holt states that in nearly all cases the children suffering from this are over seven years of age.

**Pathology.**—1. *Chronic Diffuse Nephritis With Exudation.*—Enlargement of the kidneys occurs, the surface being smooth or slightly nodular. The enlargement may be so great that the organ attains twice the normal size, and the capsule can easily be separated from the kidney itself. The color, not only of the external but of the cut surface also, is yellowish white. Considerable tumefaction of the cortex is found on section. Microscopic examination will show the epithelium to be swollen, while degeneration of a granular or fatty character follows. The convoluted tubes are dilated and thickened, while their lumen

contains broken-down granulated epithelium and cast matter. In some cases atrophy of the tubes occurs. The glomeruli will often be found compressed and atrophied from an excessive formation of new connective tissue. A fatty degeneration of the tubular epithelium is sometimes seen.

2. *In the condition known as waxy degeneration* there is considerable enlargement of the kidneys, the organs being grayish in color, translucent, and glistening. Their consistency is sometimes described as doughy. Amyloid deposits occur along the renal vessels and in the vascular tufts of the glomeruli. This process may progress until the whole organ is infiltrated, the true renal structure undergoing an atrophic degeneration. The amyloid degeneration is usually associated with the same condition in other organs, especially the liver and spleen, and occasionally in the intestinal villi. The situation of the portions of the kidneys affected by the amyloid change can be demonstrated by the iodine and sulphuric acid reaction. This consists in brushing over a section of the affected kidney a solution of iodine with iodide of potassium in water. This will give a reaction of a mahogany color. If, now, diluted sulphuric acid is applied, the color of the cut surface will change to a bluish-violet tint. The aniline violet test consists in brushing over the kidney a 1 per cent. solution of aniline violet. That portion of the kidney which has undergone amyloid degeneration will show a red or pink reaction, while the unchanged tissues are stained blue.

3. *Chronic Diffuse Nephritis Without Exudation.*—In this form the kidneys have undergone a true sclerosis; the whole organ is smaller than normal, the surfaces being nodular and the capsule adherent. Thinning of the cortex occurs, and the color is red or reddish gray.

The pathologic changes may be in part the same as those in the first variety, with the addition of a great increase of new connective-tissue elements. This increase is distributed in an irregular manner throughout the whole kidney structure. Dilatation of the tubules sufficient to form cysts of varying size occurs in places. At other times the tubules entirely disappear. Atrophy of the glomeruli follows unless chronic congestion has preceded the inflammation. If chronic congestion has preceded the nephritis, the glomeruli may be large and their capillaries dilated; generally, however, they will be seen to have undergone atrophy.

**Symptoms.**—1. *Chronic Nephritis With Exudation.*—In many cases this form of nephritis will not be recognized until there appears a slight puffiness under the eyes or occasionally dropsy in some other part

of the body. The patient will usually, on examination, be found to have had at some previous time an attack of acute renal inflammation which has never entirely subsided, but which has been unrecognized. The period of intermission since the original attack may have extended for a varying time: possibly a few months or a year or two. In some cases the symptoms of dropsy and anemia follow immediately an acute attack. As the disease progresses various digestive disturbances are



FIG. 61.—CHRONIC PARENCHYMATOUS NEPHRITIS WITH ANASARCA. The patient died in uremic convulsions three days after the photograph was taken.—(*Samaritan Hospital.*)

noticed: there may be vomiting, not only after eating, but also when the stomach is empty. The appetite is generally lessened. The bowels are very frequently constipated, although such patients often have short attacks of diarrhea. Anemia is always a prominent symptom. With each exacerbation of the disease various nervous symptoms appear. The patient will complain of violent attacks of headache, neuralgia, sometimes insomnia, and great loss of strength. This general group of symptoms is very common, and appears for a certain length of time, and

then not infrequently disappears quite suddenly, the patient in the interval becoming quite comfortable, gaining strength to a certain degree. With each recurrence the symptoms become more marked. Finally dyspnea will develop, the heart becomes irritable, vomiting increases, apparently without cause, and the patient complains of vertigo and sometimes of defects in vision. The dropsy, which has previously been slight, may extend over the whole body. (See Figure 61.) Effusions occur into the serous cavities, and the patient may die from pulmonary edema. During the exacerbations of the disease slight attacks of epistaxis not infrequently occur. During each onset the urine is scanty and highly colored, containing casts, in character granular, epithelial, and sometimes fatty or hyaline. Oil globules are sometimes found. The specific gravity of the urine is low, usually not over 1012 to 1015. The quantity of albumin will vary considerably; between the periods of exacerbation it may be quite small in amount. No matter how well the patient may seem to be between the attacks, some albumin and some tube-casts are almost always present. The urine will, however, be passed in much larger quantities during these intervening periods, but the specific gravity is never so high as that of normal urine.

The amount of urea excreted is below the normal. A certain amount of vesical irritation is quite commonly found. The duration of this form of nephritis differs according to the surroundings of the patient and the amount of kidney tissue affected.

2. *Waxy or amyloid degeneration of the kidneys* is usually accompanied by or associated with the same sort of change in other organs, particularly the liver, spleen, and intestinal canal. Ascites is a more marked symptom in this form. The urine is generally increased in amount, is yellow in color, of low specific gravity, and will contain albumin and hyaline casts, and later waxy casts. A profuse watery diarrhea is present, and is particularly marked when the amyloid changes affect the intestinal canal, which makes the prognosis extremely grave. The peculiar whiteness of the skin, known as "alabaster cachexia," is often present.

In both the preceding forms of renal disease death most commonly occurs from acute uremia, pneumonia, pericarditis or endocarditis, or from pulmonary edema or pleurisy; rarely, however, from uremia in the amyloid variety.

3. *The symptoms of chronic interstitial nephritis* in children are the same as in adults. The urine is pale in color and large in quantity. The specific gravity is very low, usually between 1002 and 1010. Albu-

min will be found in very small quantities; frequently it is not present at all for periods of varying length, as are also hyaline casts. Reduction in amount of urea is a grave symptom. Dropsy is rare at first. On the other hand, the arterial tension is generally high, as shown in the blood-pressure, and hypertrophy of the left ventricle is usually present. Atheroma of the arteries may be found even in a child as young as six years of age (Dickinson). Nervous phenomena, such as headaches, neuralgia, various disturbances of vision, and dyspnea, are very commonly seen. Death usually occurs from acute uremia, although hemorrhages, especially cerebral hemorrhages, may occur late in the disease.

The **diagnosis** of chronic nephritis in children is based practically on the same facts as similar disease occurring in adults. In cases where there are convulsions, with frequent or persistent headaches, or such conditions as anemia, cardiac hypertrophy, especially with high arterial tension, and in cases of general malnutrition, the urine should be frequently and carefully examined. When any of the group of symptoms pointing to renal disease manifest themselves, the patient should be kept under observation for a considerable period, and the case carefully studied.

**Prognosis.**—The outlook for complete recovery in any of these forms of nephritis is not favorable. On the other hand, much can be done in the first variety for the comfort of patients and the prolongation of their lives. There is no doubt that many cases affected with chronic nephritis, especially the exudative variety, live for years, providing they are placed amid comfortable surroundings, are kept absolutely free from worry, and in a climate of reasonably equable temperature, particularly one free from extremes of cold and heat. The prognosis in cases of waxy kidney is about the same as in the preceding form. It is possible that recovery may take place when, in cases resulting from prolonged suppuration of bone, the diseased structure has been removed. The prognosis in the interstitial variety of nephritis is always bad, although the progress of the disease is generally quite slow. The immediate prognosis will depend considerably on the amount of dropsy, the existence of valvular disease of the heart, the amount of urea excreted, and the strength of the general excretory power of the kidneys.

**Treatment.**—Children affected with chronic nephritis should be placed amid surroundings free from nervous worry and strain of all kinds. It is very important that such children, as soon as the disease is recognized, be kept from school, or at least from hard study. If possible, they should be sent to a dry, warm climate, especially during

the winter months. Great care should be exercised that these patients do not take cold. Woolen underclothing should be worn next to the skin at all seasons of the year. While overfatigue is extremely dangerous, yet regular exercise in the open air is of the greatest benefit. They should be dressed warmly in winter, and in summer their clothing should be so regulated as to allow them as much coolness as possible, yet avoiding any danger of chilling. The general aim of the treatment is to retard the progress of the disease as much as possible, and when symptoms arrive, to relieve them. Tonics are nearly always indicated. A good rule to remember in the administration of remedies to these patients is that during the periods of quiescence of the disease as little medicine should be given as possible. While milk is in many respects the best article of diet, yet frequently it cannot be borne for a long period of time; it becomes extremely disgusting to some patients, and will finally do more harm than good. Sometimes, when milk cannot be taken by itself, it can be used on desserts, on fruit, or given in some other way to make it more palatable. Much meat must not be allowed, especially where there is a tendency to a diminution in the excretion of urea, and salt meats should be prohibited. The lighter soups, fresh fish, oysters, and foods of this description may be given in moderation, as also may farinaceous foods and the starchy vegetables. The patient should be encouraged to drink water, and for this purpose many of the mineral waters are recommended, not so much for their own inherent qualities as on account of their being more palatable.

Iron should be prescribed when anemia is a prominent symptom, and when considerable dropsy exists, diuretics, saline laxatives, and calomel in small doses are needed. When the heart is weak, this condition will naturally call for cardiac stimulants. The skin should be made to do as much work as possible, thereby easing the crippled kidneys, and with this in view sponge-baths of hot water or occasional vapor baths are useful. Attacks of uremia should be treated in the usual way. If the arterial tension is high and there are convulsions or stupor, blood-letting may be resorted to. In many of these cases nitroglycerin will also be found useful. The hot pack, the electric hot-air bath and the alcohol vapor bath before alluded to are probably the best means of producing rapid diaphoresis. In cases where the uremic convulsions are marked and are accompanied by dilatation of the pupil, morphin may be administered hypodermically.

Cortical decapsulation of the kidney (Edebohl's operation) may be resorted to in certain cases. There is a considerable diversity of opinion concerning its value; but our experience would lead us to recommend

it in cases that are steadily progressing toward a fatal termination despite ideal medical treatment. If the specific gravity and the urea excretion are constantly falling, the casts are numerous, and uremia seems impending, we advise that the Edebohls' procedure be followed.

### PERINEPHRITIS

**Definition.**—Perinephritis consists of an inflammation of the connective tissue surrounding the kidney.

**Etiology.**—In origin it may be primary or secondary. If primary, the cause may be from cold or exposure (?) or traumatism. Occasionally it develops without any known cause. Secondary perinephritis may follow suppurative diseases of the kidney, a perforative appendicitis, or even caries of the vertebræ.

**Pathology.**—The perinephric tissues of both kidneys are affected with equal frequency, and the disease is as common in girls as in boys. It may be found at any age. When the inflammatory process progresses to the formation of an abscess, the latter usually burrows between the lumbar muscles, and may appear superficially in the posterior part of the body, near the middle of the ileocostal space. Sometimes it may proceed between the abdominal muscles and point above Poupart's ligament. Occasionally it may appear at the upper and inner aspect of the thigh, or it may rupture into the peritoneal cavity, thoracic cavity, vagina, or bladder.

**Symptoms.**—The symptoms of perinephritis are those of acute inflammation. The attack usually begins with a chill, fever, and pain. The pain is usually felt in the lumbar region, in the groin; along the inner side of the thigh, and in the knee, and is generally increased on moving the leg. When the disease has existed for some time, a distinct tumor, accompanied by tenderness, may be seen and felt over the region of the kidney on the affected side, and this tenderness may extend to the hip or along the back. As the inflammation proceeds there may be stiffness in the hip on the affected side. The thigh is flexed and extension will cause resistance and pain. Other movements of the limb, however, are normal. Symptoms referable to the kidneys themselves are not present in all cases. Their presence will depend, to a certain extent, on whether or not these organs share in the inflammation. Sometimes there may be some pain on micturition, and this may be increased in frequency. When pyelitis exists, the urine will contain pus. The disease may vary greatly as to the length of its duration.

Acute cases may run a course of from four to eight weeks. The disease may, however, last for several months.

**Diagnosis.**—The disease with which perinephritis is most likely to be confounded is *inflammation of the hip-joint*. The two affections, however, have distinct points of difference which will make the diagnosis one of not great difficulty. Perinephritis is a disease of much more intense and rapid onset, and the general symptoms are those of an acute inflammation; whereas in hip-joint disease the condition develops slowly and the constitutional symptoms may be wholly absent during the early stages of the affection. In perinephritis there is interference with flexion and extension of the thigh, but the other motions are not interfered with; whereas in hip-joint disease all movements of the joint are restricted, and there is also tenderness in the joint itself, and pain is frequently referred to the inner aspect of the knee. The characteristic secondary changes in the thigh, and particularly the atrophy of the buttock (*glutei*), which are always present in hip-joint disease are absent in perinephritis. *Psoas abscess* may be mistaken for perinephritis, but in the former we usually have present the symptoms of tubercular disease, its characteristic temperature, and very possibly some deformity of the spine may be seen to aid in the diagnosis.

**Prognosis** is fairly good. The majority of cases recover from this disease.

**Treatment.**—This should consist in complete rest, and when the case is seen early, counterirritation should be applied over the lumbar region, or, in some cases, an ice-bag may give a better result than heat. Poultices sometimes give relief. As soon as the presence of pus can be demonstrated, an incision should be made and free drainage established.

### PYELITIS

Pyelitis is an inflammation of the mucous membrane lining the pelvis of the kidney. When portions of the true kidney structure are involved, the condition is known as pyelonephritis. When an accumulation of pus exists in the pelvis of the kidney, the condition is known as pyonephrosis.

**Etiology.**—The condition may arise from congenital malformations of the kidneys or ureters, from tuberculosis, or from new growths. It may also be caused by an extension of an inflammation of the surrounding tissues. Extension upward of a septic inflammation of the genito-urinary tract is a very common cause. Rarely it arises from an irritation produced by a renal calculus. Most of these ascending in-

fections are due to the colon bacillus, and in most cases they originate from cystitis in female infants. (See Cystitis.) Urinary infection is not always proved, it may arise from contiguity or through the blood stream. An acute form of the disease may follow an attack of the infectious fevers or of septicemia.

**Pathology.**—Pyelitis may affect one or both kidneys. In the acute form the mucous membrane presents the usual appearance of acute catarrhal inflammation; it is congested, swollen, and, in the severer cases, with formation of pus, small hemorrhages will be found. When the disease becomes chronic, the mucous membrane is thickened and granular.

**Symptoms.**—The symptoms will, to a certain extent, depend upon the cause. When the attack is acute and is produced by a stone or other cause of direct renal irritation, there may be chills, pain, fever, and attacks of renal colic. When the cause is tuberculosis or abscess in the kidney, there will be recurring chills, fever, sweats, and some pain. The patient will suffer progressive loss of flesh, and present the general appearance of deteriorated health. Unexplained fever in baby girls should always suggest the possibility of pyelo-cystitis. Such babies exhibit pallor and fever (usually of intermittent type). Fever may be sustained or absent, however. The only safe rule is to examine the urine. The quantity of urine is usually somewhat diminished. Its reaction is acid; albumin, pus, and epithelial and blood-cells are found in it. Hyaline and granular epithelial casts may be present, Bacteria are usually demonstrable.

The **diagnosis** can be made from the symptoms and from examination of the urine. In differentiating this condition from cystitis it should be remembered that the urine is less apt to be acid in reaction in inflammation of the bladder, and in the latter condition the quantity of pus will be much less. The absence of tube-casts and of renal epithelial cells, as well as the more severe general symptoms, will generally be sufficient to differentiate it from nephritis.

**Treatment.**—The patient should be placed on a fluid diet, and when the urine is irritating, by reason of its high degree of acidity, the latter should be neutralized by the administration of citrate of potassium or by free administration of a simple alkaline water. Urotropin, carefully administered, may render splendid service. Counterirritation over the lumbar region by the use of dry cups, poultices, or mustard plasters should be employed. When pyonephrosis exists, the question of surgical interference must be considered. If only one kidney is affected, its removal promises a chance of recovery; but usually both

organs are involved. Bacterins sometimes yield good results when alkalies and urotropin have failed.

### RENAL CALCULI

Renal calculi may be formed at any period of life. The stones may vary in size very considerably. They are usually found in the pelvis and calices of the kidney and are composed of uric acid. When very small, they may be seen as small granular deposits in the pelvis of the kidney.

**Symptoms.**—When the deposits are small, they may be excreted through the pelvis of the kidney and ureter without producing any symptoms. When large, however, their passage generally causes the condition known as renal colic. The patient is suddenly seized with intense pain and tenderness over the affected kidney. The pain soon radiates around the affected side diagonally across the abdomen to the region of the bladder. There may be pain in the perineum, and in boys the testicle on the affected side is retracted. Sometimes the pain may even radiate to the opposite side of the body. The attack will continue at intervals until the stone has reached the bladder, when it usually ceases suddenly. Marked tenderness may exist over the affected kidney (see above). During the attack, and for a short time afterward, the urine may contain traces of blood and some albumin. It is to be recalled, however, that the kidneys act alternately and that such urinary finds may be noted at one time and absent at another. When pyelitis has been produced by the irritation of the stone, the symptoms of this condition will be present, and the urine will contain pus and epithelial cells from the pelvis of the kidney. In some cases the pain is so great as to produce collapse. When the stone becomes impacted in the upper part of the ureter, hydronephrosis or pyonephrosis occurs.

The **treatment** of renal calculi in children is the same as in the adult.

### TUBERCULOSIS OF THE KIDNEY

Tuberculous disease of the kidney usually occurs as secondary complications of general tuberculous disease. The source of infection is often in the blood, and very rarely the extension of tuberculous disease from the bladder. It is stated that the disease generally begins in the pelvis and calices of the kidney. Later the pyramids and cortex become involved. As a rule, only one of the kidneys is affected. Not infrequently a tuberculous perinephric abscess will coexist.

**Symptoms.**—These, in many cases, are obscure. Pain or tenderness in the region of the kidney and possibly some swelling are common features; the latter is particularly the case if perinephritis exists. Again, we would call attention to the “kidney triangles.” Irritability of the bladder is generally present. The urine is decreased in amount and contains pus. A sure point of diagnosis is the recognition of the tubercle bacillus in the urine. Renal tuberculosis is most commonly seen in children between two and twelve years of age, although it may be found at any period of life.

The **treatment** is purely surgical, and consists in removing the kidney.

### TUMORS OF THE KIDNEY

Tumors of the kidney in childhood may be benign or malignant. The former are very rare. Aldibert has reported three cases: one each of adenoma, fibroma, and fibrocystic tumor. These tumors can be recognized by their slow growth and by their mild constitutional symptoms. The commonest forms of malignant tumors of the kidney occurring in children are sarcoma and carcinoma, the former being most frequently seen. They are usually primary growths, and rarely occur in children under five years of age. After this time, however, they are by no means a rare form of abdominal neoplasm. They may be congenital, however.

**Pathology.**—The type of sarcoma is usually round- or spindle-celled, but myosarcoma is sometimes seen. Endothelioma is a rare form that one of us has seen. The child was operated upon; but metastases occurred in an astonishingly short time. Tumors may grow from the cortex or the pelvis of the kidney, and sometimes from the adrenals. Infiltration of the whole organ may take place even to such an extent as to destroy the entire renal structure. Metastasis may occur in the opposite kidney, lungs, or other neighboring structures. Hydronephrosis, due to pressure upon the ureter, or serious complications, such as thrombosis, from pressure upon the vena cava, may take place. As the tumor grows it becomes adherent to the surrounding organs. Ascites and general peritonitis may appear in the later stages. In size the tumor may reach very considerable proportions. According to Holt, the weight may be as high as fifteen pounds, and he states that one case has been reported by Jacobi in which the tumor weighed thirty-six pounds. The right kidney is rather more frequently involved than the left.

**Symptoms.**—The principal symptoms are a rapidly growing tumor, with progressive emaciation and cachexia. Pain may be present, but not infrequently it is slight or may be entirely absent. The tumor may at times be made out over the region of the kidney, but oftener, as it grows in the direction of least resistance, is felt underlying the large bowel. The increase in size is rapid. The urine may at intervals contain blood, pus, or albumin. Sometimes this does not occur until late in the disease.

The **prognosis** is unfavorable.

The **treatment** should be by operative means. (See Abdominal Tumors.)

## CHAPTER XV

### DISEASES OF THE GENITAL ORGANS

#### ADHERENT PREPUCE AND PHIMOSIS

Adhesions between the prepuce and the glans penis are not infrequent at birth. They may appear in congenital forms or may be acquired. If the latter, they are usually caused by an irritation set up by the collection of smegma accumulating under a long, tight foreskin. Phimosis is that condition by which the foreskin is prevented from being drawn back over the glans by an abnormal smallness of the opening of the prepuce. Both adherent prepuce and phimosis of a not marked degree are seen in a large number of children, and both conditions frequently disappear shortly after birth. They may, however, give rise to quite a variety of very distressing **symptoms** and **sequelæ**. Thus we may have great pain on urination, which will cause the infant to cry vehemently, and in many of these cases it will be seen that the foreskin will balloon during the act of urination, showing that the prepuce is adherent to the glans, the stream of urine being very small, or in some cases simply dropping away. A certain amount of urine is generally retained and will increase the irritation—in fact, may set up an actual balanitis or even a cystitis. Stricture of the meatus may also be produced. Various nervous phenomena may accompany the condition. In infants as well as older children the symptoms may be very varied. Choreic movements, incontinence of urine, convulsions, and persistent spasm of certain sets of muscles may be produced, and a chronic state of malnutrition often follows this condition. There may be even a prolapse of the rectum, or hernia from constant straining in voiding the urine. Adherent prepuce and phimosis are undoubtedly among the most common reflex causes of masturbation in young boys.

The **treatment** should consist in breaking up the adhesions by passing the end of a blunt probe between the mucous membrane and the glans and dilating the preputial orifice with the blades of a pair of surgical dressing forceps. After this is done the prepuce should be drawn back so as to expose the glans, which should then be cleansed and anointed with vaselin or any bland antiseptic ointment, after which the prepuce should be brought forward again. This should be done

gently and repeated every day until danger of readhesion be passed. It is our experience—and we habitually give attention to the condition of the foreskin in all male babies—that the careful and prompt operation of stripping is in the majority of cases sufficient. The need for the operation of circumcision is much exaggerated. It should be borne in mind that when the foreskin is kept back too long there is some danger of producing the condition known as paraphimosis. Where dilatation does not produce relief after several repetitions, the foreskin may be slit up as far as the corona glandis, along the dorsum, and the edges trimmed off with a pair of scissors. The mucous membrane may be stitched to the skin with fine silk or fine catgut. If this does not give relief, the regular operation of circumcision should be performed. It should be mentioned in this connection that adhesions of the clitoris are not uncommon. They demand similar treatment.

### PARAPHIMOSIS

Paraphimosis is a condition in which the prepuce has been drawn back over the corona glandis, and by reason of an abnormally small preputial orifice or an increased size of the glans, cannot be returned to its proper position. Following the retraction of the prepuce there are usually swelling of the glans and considerable edema. It may occur from a variety of causes. It may be congenital, although this form is rare.

The **treatment** consists in endeavoring to reduce the paraphimosis as quickly as possible by pressing the glans with the thumb and finger of one hand, while with the other hand an effort is made to draw the foreskin over the corona. Where this fails, it may be necessary in some cases to make a series of small punctures of the edematous mucous membrane, after which the same manipulation should be repeated. If the second method is not successful, the end of a blunt-pointed bistoury should be introduced under the edge of the prepuce which forms the constricting ring, and severing it on the dorsum of the glans. It may be necessary to divide the ring at more than one point. The local injection of cocain, the part having been previously anesthetized with chlorid of ethyl or ice and salt, is often all that is necessary, but occasionally etherization of the patient will be required before operating. Circumcision should not be resorted to unless necessary.

### BALANITIS

This condition is occasionally met with in children, and is frequently the result of neglected phimosis. Very considerable swelling of the

prepuce occurs, accompanied by discharge, often of large quantities of pus, producing great pain and scalding on micturition. The discharge is usually very offensive, the characteristic odor of smegma possessing almost pathognomonic significance.

The **treatment** should consist in syringing the cavity beneath the prepuce with a warm, bland antiseptic solution. After relief of the phimosis, application of lead-water will often reduce the inflammation.

### EPISPADIAS

In males this name is applied to the malformation in which the urethra opens on the dorsum of the penis. It is very commonly associated with extrophy of the bladder. Occasionally there is a defect in the union of the anterior abdominal wall and a cleft in the symphysis pubis. In female children the anterior wall of the urethra is absent. The nymphæ and clitoris are generally split.

The **treatment** should be surgical.

### HYPOSPADIAS

The condition known as hypospadias is the result of arrested development in the urethra and corpus spongiosum. Normally, the urethral groove should, by the uniting of its sides, be converted into a canal. This process of union begins at the base and extends to the end of the penis, and arrest of this process of development may cause the urethra to open at any point along the inferior margin of the penis. In female children the urethra usually opens directly into the vestibule.

### VULVOVAGINITIS

Vulvovaginitis in children is usually found in two forms, the catarrhal and the gonorrheal. The catarrhal form arises from irritation, which is produced by a variety of causes. It is usually a mere vulvitis and not a vulvovaginitis. In very young children it may be brought about by the continued use of diapers soiled with discharges or from general lack of cleanliness, and accumulation of smegma. Seat-worms are a common cause. It may also be due to traumatism and attempts at rape. A form of vulvovaginitis known as the aphthous variety is found in ill-nourished and unhealthy children, or may occur as a sequela of one of the continued fevers or any constitutional disease, as nephritis, tuberculosis, etc. Usually, however, when true vulvo-

vaginitis occurs, the organism of Neisser (gonococcus) will be found in the discharge. Edward Martin has shown that such infections may take place in many ways. The organisms have been recovered in scrapings from door-knobs, etc. Other organisms have been found in vaginal discharges, the diplococcus pneumoniae and various pyogenicocci being the principal ones.

**Pathology.**—This does not differ from the pathology of similar conditions in the adult except that there is less tendency to invasion of the uterus, tubes, peritoneum, etc. The cervix and urethra are involved, however.

In girls at the age of puberty it sometimes arises from retention of the menstrual secretion by an imperforate hymen.

The **symptoms** are those found in other forms of catarrhal inflammation. There are, generally, discomfort, some swelling of the parts, a burning pain on micturition, and a local rise of temperature. The secretion is at first arrested, the parts becoming dry, but later it is considerably increased, often with formation of pus. The patient complains, as a rule, of continual smarting and burning. There may be, in the early stages, some general rise of temperature.

In the *gonorrheal form* the symptoms are the same as in the previous variety, except that they are apt to be more intense. The discharge is free, and consists of thick, greenish-yellow pus, in which gonococci can be found. The parts are excoriated, and generally very painful. Some swelling of the inguinal glands may occur. It is by no means uncommon in children, as in adults, to find the urethra involved in the general irritation. A positive differential diagnosis, however, cannot always be made without the aid of the microscope. It should not be forgotten that the process may extend, as in the adult, to the peritoneal cavity. (See Acute Peritonitis.)

The **prognosis** in both forms of vulvovaginitis is good. However, it must be borne in mind that in the gonorrheal form the infection may spread into the urethra and bladder, uterus, tubes, peritoneum, etc., producing inflammation of these parts. Such complications are relatively very rare. Under the best conditions, however, the course of the disease is usually a long one. As in the adult, gonococci may remain latent in the genital tract for a long time.

The **treatment** consists of absolute cleanliness. The parts should be bathed in warm water and Castile soap, after which they may be dusted with a powder consisting of calomel and starch, bismuth and starch, or boric powder. Ichthyol, in the proportion of 5 parts to 100 of glycerin, may be applied on a tampon, or pledgets of cotton saturated

in lead-water and laudanum may be used. Where the irritation is caused by worms, rectal injections of an infusion of quassia in the strength of one to two ounces to a pint of water may be used with benefit. The aphthous form should be treated not only by local applications, but also by attention to the general health, efforts being made to build the patient up, so far as possible, by tonics and nutritious food.

Gonorrheal vulvovaginitis is best treated by local applications of corrosive sublimate, 1 : 5000; boric acid, one dram to one pint; creolin, 1 : 500, or, in some cases, a 2 per cent. solution of nitrate of silver. Some of the organic silver salts (protargol and argyrol) have proved of signal value in these cases. Brewer's yeast, locally applied, has also yielded excellent results. These should be applied by means of pledgets of cotton placed between the labia, or may be injected in the vagina by means of a small rubber catheter attached to a fountain syringe. It is our custom to use a silver-salt (in proper strength) solution once daily, and a thorough irrigation with potassium permanganate solution three times daily. When the symptoms are declining rapidly, we know of no astringent more valuable than the sulphocarbolate of zinc (gr. iii to gr. v to  $\mathfrak{J}$ i). The cervix uteri and urethra demand special attention in intractable cases. Before applying any of the above, it is generally well to wash the parts thoroughly with warm water and Castile soap, after which they should be carefully dried by means of pieces of absorbent cotton. The parts may be dusted with a powder consisting of bismuth and starch or pulverized oxid of zinc and boric acid. Bacterins, particularly autogenous bacterins, have proved of much service in the treatment of gonorrheal vulvovaginitis.

## ORCHITIS

Inflammation of the testis is seldom seen in childhood, except as the result of traumatism. It may rarely follow an attack of mumps, but in childhood this is much less frequent than in the adult. Not infrequently orchitis is accompanied by hydrocele.

The **treatment** should consist in supporting the testicle by means of a suitable bandage or pads of cotton and the local application of lead-water and laudanum. Laxatives should be used to keep the bowels freely opened.

## TUBERCULAR DISEASE OF THE TESTICLE

Tubercular disease of the testicle is rarer in infancy and childhood than in adult life. When present, the testicle will be found considerably

swollen, nodular, and not very tender. As the disease progresses adhesions may form between the testicle and the scrotum. The glands may break down late in the disease and suppuration occur. Tubercular disease of the testicle is found in two forms: (1) Secondary to a general tubercular infection or (2) as a part of a localized tuberculosis of the genito-urinary tract.

**Treatment.**—Where the diseased condition of the testicles is a part of a general tuberculosis, the orchitis should be treated symptomatically and attention paid to the general tubercular infection, the treatment of which is considered in the chapter on Tuberculosis. In all cases of tubercular disease of the testes castration should be performed; especially should this be done to young children. There is always a danger of general systemic infection resulting from the diseased testicle.

### EPIDIDYMITIS

Inflammation of the epididymis may be caused by traumatism or by continuation of irritation of the urethral mucous membrane. The epididymis will be found considerably enlarged and very tender, and by its swelling it will push the testicle forward. The spermatic cord is often inflamed, enlarged, and extremely painful on pressure. It is not uncommon to find the whole scrotum swollen and very painful.

The **treatment** should consist of absolute rest, the patient lying on his back. The bowels should be kept open. Local applications in the form of hot poultices or lead-water and laudanum should be made to the scrotum. The scrotum should always be supported in this as in all forms of inflammation of this region. Rotch recommends that the testicle be always placed in such a position that the lower end of the gland points upward.

### HYDROCELE

This condition is not at all uncommon in children, being quite frequently met with in the early years of life, and may be acute or chronic. It may result from a variety of causes, sometimes arising from traumatism, as by pressure, and sometimes during or after attacks of influenza, typhoid fever, scarlatina, or mumps. Some cases are tuberculous, (Babcock.) It may in some instances result from simple irritation. Occasionally it is congenital. The form known as infantile hydrocele is really a condition where the tunica vaginalis and funicular process are distended with fluid, the processes being closed at the internal abdominal ring. The funicular part of

the process may remain open and be shut off from the tunica vaginalis.

In the third variety there may be an encysted hydrocele of the cord, due to distention of an unclosed segment of the funicular process.

**Diagnosis.**—The condition with which it is most likely to be confused is scrotal hernia, the diagnosis of which has been given under the head of Hernia.

**Treatment.**—In acute hydrocele rest and the local application of cold, ichthyol, or lead-water cloths are employed, or treatment by puncture if the above be unsuccessful. Chronic cases should be treated by a truss in the same manner as hernia, an effort being made to close the neck of the canal.

In all forms of irreducible hydrocele the treatment is by evacuation of the fluid by means of a small trocar and cannula under antiseptic precautions. If this is unsuccessful, *it is advisable that the sac be extirpated* or a weak solution of iodine be injected. Cures have resulted from the local injection of tuberculin. When a cure is not effected by means of simple evacuation, the sac should be laid open, the cavity packed with iodoform gauze, the strictest antiseptic precautions being followed, and the wound allowed to heal by granulation. If the sac be large, a small portion of its walls on each side of the incision should be cut away. This is a perfectly safe and rapid method of radical cure of this condition. Many cases of the infantile variety get well without treatment.

*Hydrocele in Female Children.*—Although hydrocele is much rarer in female than in male children, yet it sometimes occurs. It consists in a collection of fluid in the tube-like pouch of the peritoneum, which accompanies the round ligament through the inguinal canal and is known by the name of the canal of Nuck. Occasionally the exudation of fluid may take place in the tissues of the round ligament itself or in the labium major, external to the covering of the round ligament.

The **symptoms** will be the appearance of a tumor in the labium or the inguinal region. Fluctuation will be obtained in this tumor. It will also be translucent. Appearing in girls at the age of puberty, it might possibly be confounded with a cyst or abscess of the vulvovaginal gland, but the lack of inflammatory symptoms and the fact that cysts are found in the upper and outer part of the labium major would aid in the diagnosis. Pudendal hernia would be excluded by the fact that in hydrocele there is no impulse on coughing and the other symptoms of hernia are absent.

The **treatment** is the same as that of hydrocele in male children.

VARICOCELE is a very rare affection in childhood. Ashby and Wright claim they have never seen it earlier than the tenth year.

**Treatment** will be the same as that for the same affection in adults.

### UNDESCENDED TESTICLE

Ordinarily the testes descend into the scrotum during the eighth month of intra-uterine life, but occasionally children are born with the glands still within the abdominal ring. In cases where some years have elapsed without the descent of the testicles it is quite possible that atrophy may take place, although this is by no means necessary. It sometimes happens that one or both of the testicles will descend and return to the abdominal cavity again. It is a common thing to find the testicle in a baby or young child as high as the external ring of the inguinal canal. This should not be mistaken for an undescended testicle.

**Treatment.**—The location of the gland is most important in determining the treatment. In cases where the testicle shows a tendency to descend it should be allowed to do so, as this may take place even after a lapse of several years.

The majority of cases, however, will require surgical interference. For the details of such operation the reader is referred to surgical works upon the subject.

### TORSION OF THE SPERMATIC CORD

Occasionally one of the testes is twisted upon the cord sufficiently to cause constriction of the circulation, and even gangrene. This generally happens in an undescended or partially descended testicle.

It is very difficult to differentiate between this condition and a strangulated hernia.


Operation is necessary; the cord untwisted if possible, or, if there be any doubt as to the vitality of the tissues, the testicle and cord must be excised.









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